THE PSYCHOLOGICAL IMPACT OF MOTOR VEHICLE ACCIDENTS

by

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I certify that this thesis contains no material which has been accepted for a degree or diploma by the University or any other institution, except by way of background information where acknowledgment is made in the text of the thesis, and that to the best of my knowledge and belief this thesis contains no material previously published or written by another person except where due acknowledgment is made in the text of the thesis.

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ABSTRACT

Exposure to a traumatic event may result in the development of Posttraumatic Stress Disorder (PTSD) or Acute Stress Disorder (ASD). Biopsychosocial variables associated with these disorders following motor vehicle accident [MVA] trauma were the subject of this investigation. Reviews of the diagnostic classification of the psychological sequelae of trauma exposure, and theoretical models of the aetiology of posttraumatic stress disorders preceded the empirical studies. Posttraumatic responses were concluded to be affected by multiple biopsychosocial mechanisms best represented by an integrated aetiological model. Consequently, the need for multimodal assessment of posttraumatic psychological responses was evident, and existing assessment methods were discussed. ASD was found to be the subject of relatively little research to date when compared with PTSD, highlighting a need for comprehensive examination of the more recently introduced diagnostic entity.

The empirical studies focused on the examination of multi-variable profiles associated with diagnosis-specific psychological sequelae to MVA trauma. The first study was a large scale screen of an Australian university student sample (N = 425), and was conducted to investigate MVA trauma exposure and associated posttraumatic symptoms. In the second study, psychometric data were used to investigate coping styles and belief systems associated with the development of PTSD, ASD and subclinical responses to MVA trauma (N = 83). The results indicated little difference in the profiles of the ASD and subclinical groups, which were characterized by adaptive coping and rational belief

systems. The PTSD group profile was characterized by a combination of adaptive and maladaptive coping, and no differences were found between the three groups in terms of rationality of beliefs. Study three demonstrated the use of a multimodal tool to assess associations between recollections of peritraumatic responses and posttraumatic diagnostic outcomes. Psychological and psychophysiological reactivity to trauma-related and neutral idiosyncratic imaged events were examined using a four stage guided imagery methodology (N = 51), and multimodal group-specific response patterns were detected. Study four investigated perceived posttraumatic psychological outcomes of the three experimental groups (N = 83). The ASD and subclinical group profiles reflected adaptive and positive posttraumatic recovery, but also reflected that all participants, regardless of diagnosis, were psychologically affected by MVA exposure. The PTSD group profile was characterized by a broad range of negative posttraumatic outcomes, pervasive in many aspects of functioning. Consideration was given to factors that may have led to these between group differences.

The results of the empirical studies supported the proposition that PTSD and ASD are distinct diagnostic entities that may be differentiated on the basis of a complex array of biopsychosocial variables. The implications of the results for the assessment, diagnosis and treatment of posttraumatic responses were discussed, and directions for future research were suggested.

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CHAPTER ONE

INTRODUCTION TO THE INVESTIGATION

1.1 Definition of the problem

Psychological trauma has been described as the most severe and potentially incapacitating form of human stress (Everly, 1995a). Exposure to a traumatic event may result in the development of a psychiatric disorder (e.g., McFarlane & de Girolamo, 1996). A traumatic event has been defined in the fourth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) as "...an event during which an individual is exposed to actual or threatened death or serious injury, or a threat to the physical integrity of self or others" (American Psychiatric Association [APA], 1994, pp. 427-8). In order to meet the DSM-IV criteria for a traumatic event, the individual's psychological response to the event must have involved intense fear, helplessness or horror.

Formal psychiatric diagnoses of posttraumatic stress symptoms have been used to classify the severity and nature of posttraumatic psychological responses (e.g., APA, 1994; Jensen & Hoagwood, 1997). The conceptualization of posttraumatic responses as psychiatric entities has been demonstrated by the inclusion of Posttraumatic Stress Disorder (PTSD) and Acute Stress Disorder (ASD) in the DSM-IV (APA, 1994). PTSD and ASD are psychiatric disorders characterized by a range of symptom clusters including reexperiencing symptoms, avoidance and numbing symptoms, and increased physiological arousal (e.g., King, King, Foy, & Gudanowski, 1996). The ASD diagnosis includes a dissociative symptom cluster. The symptom clusters for PTSD and ASD are not trauma-specific and, therefore, apply to all types of trauma exposure.

PTSD has been acknowledged as a diagnostic entity since 1980 (APA, 1980) and ASD was introduced in the DSM-IV (APA, 1994). The introduction of

ASD as a diagnostic entity formally endorsed that posttraumatic psychological response is not an 'all-or-none' dichotomy. The introduction of the ASD diagnosis provided recognition that individuals exposed to trauma may experience significant short term posttraumatic symptoms, and acknowledged the relevance of dissociative symptoms in the framework of posttraumatic responses (e.g., Blanchard, Hickling, Vollmer & Loos, 1995; Bryant & Harvey, 1999).

The present investigation has two main objectives. The first is to produce multi-variable psychological profiles associated with the development of PTSD, ASD and subclinical posttraumatic responses. This process is proposed to contribute to the body of knowledge of the development of diagnostically distinct psychological responses to trauma, and to investigate variables associated with posttraumatic adjustment following ASD. The second objective is to specifically focus on the psychological impact of motor vehicle accident [MVA] trauma. MVA trauma has been found to be a frequently occurring trauma type that may result in decreased quality of life and compromised mental health (e.g., Blanchard & Hickling, 1997; Cagnetta & Cicognani, 1999; Dougall, Ursano, Posluszny, Fullerton, & Baum, 2001; Fectau & Nicki, 1999; Hickling & Blanchard, 1999; Koren, Arnon, & Klein, 1999; Lowenstein, 2001).

A considerable amount of literature has focused on trauma associated with war (e.g., Aldwin, Levenson, & Spiro III, 1994; Beckham, Crawford, & Feldman, 1998; Beckham, Feldman, & Kirby, 1998; Blanchard, 1990; Blanchard, Kolb, Pallmeyer, & Gerardi, 1982; Brennan, 1998; Fairbank & Keane, 1982; Fontana & Rosenheck, 1998; Gerardi, Blanchard, & Kolb, 1989; Hamilton & Workman, 1998; Keane, Fairbank, Caddell, Zimering, & Bender, 1985; Lifton, 1993; Maercker, Beauducel, & Schutzwohl, 2000; Marmar et al., 1994; Michultka,

Blanchard, & Kalous, 1998; Orr, Meyerhoff, Edwards, & Pitman, 1998; Penk, Peck, Robinowitz, Bell, & Little, 1988; Rundell & Ursano, 1996; Solomon, Laror, & McFarlane, 1996). However, the effects of trauma types such as physical and sexual assault (e.g., Boudreaux, Kilpatrick, Resnick, Best, & Saunders, 1998; Davidson, Tupler, Wilson, & Connor, 1998; Davis & Breslau, 1994; Duggan & Sroufe, 1998; Feeny, Zoellner, & Foa, 2000; Foa & Riggs, 1993; Griffing, 1998; Maker, Kemmelmeier, & Peterson, 2001; Matorin & Lynn, 1998; Morrissette, 1999; Rothbaum, Kozak, Foa, & Whitaker, 2001; Shriner, 1999; Smucker, Dancu, Foa, & Niederee, 1995; Wenninger & Heiman, 1998), natural and man-made disasters (e.g., Green, Lindy, Grace, & Leonard, 1992; Hodgkinson & Stewart, 1991; Inkelas, Loux, Bourque, Widawski, & Nguyen, 2000; Moinzadeh, 1999; Wang et al., 1999), criminal acts (e.g., Campfield & Hills, 2001; Harrison & Kinner, 1998; Janoff-Bulman, 1995; Kilpatrick & Resnick, 1993), physical illness and injury (e.g., Andrykowski & Cordova, 1998; Daviss et al., 2000; Peretz, Baider, Ever-Hadani, & De-Nour, 1994; Thompson, 1999), traumatic loss (e.g., Green et al., 2001; Polatinsky & Esprey, 2000; Walker & Davidson, 2001), and accidents (e.g., Blanchard & Hickling, 1997; Frommberger et al., 1998; Harvey & Bryant 1999a, 1999b, 1999c, 1999d; Hickling & Blanchard, 1999; Lowenstein, 2001; Margiotta, 2000; Mayou, Tyndel, & Bryant, 1997; Mirza, Bhadrinath, Goodyer, & Gilmour, 1998; Watts, 1995) have also been the focus of considerable research in civilian populations.

A review of studies investigating civilian trauma reported that lifetime exposure to a variety of traumatic events is relatively common (40-70%), with prevalence rates of PTSD ranging from 18 to 28% for individuals exposed to some type of civilian trauma (Resnick, Falsetti, Kilpatrick, & Freedy, 1996), and

prevalence rates of ASD ranging from 6 to 33% (Bryant & Harvey, 2000a). In the United States National Comorbidity Study (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995), 60% of men and 51% of women surveyed had experienced at least one traumatic event, with 8% of the sample (N = 8,098) estimated to have a lifetime diagnosis of PTSD. Statistics describing the lifetime prevalence of ASD were not found in the literature to date.

MVA trauma has been reported as a regrettably frequent trauma type, that may result in clinically significant psychological sequelae (e.g., Blanchard, Hickling, et al., 1998; Buckley, 2000; Frommberger et al., 1998; Koch & Taylor, 1995). It has been recognized that a substantial number of MVAs do not meet the criteria for a traumatic event, and despite being unpleasant experiences, have not been psychologically traumatic for the individuals involved. However, it has been proposed that dismissing the potentially traumatic nature of an MVA may be a hindrance to early intervention and appropriate mental health care (e.g., Hickling & Blanchard, 1999).

MVAs have been described as the most frequent trauma type experienced by American men (e.g., Blanchard & Hickling, 1997). Assessment of the psychological impact of MVA trauma may facilitate increased community awareness of the need for psychological support following MVA trauma, and may prevent chronic outcomes by early intervention and prognostic profiling (e.g., Bryant & Harvey, 1999). Given that the DSM-IV (APA, 1994) diagnoses of PTSD and ASD are not trauma specific, it has also been proposed that investigations of the development of these disorders following exposure to one trauma type have relevance for understanding the psychological impact of exposure to other types of traumatic events.

1.2 An overview of the investigation

The present investigation commences with theoretical chapters devoted to the diagnostic classification, aetiological models, and multimodal assessment of posttraumatic responses. These chapters summarize the current state of the literature relevant to the empirical studies, and highlight the relatively small amount of research examining ASD, when compared to the considerable wealth of research regarding PTSD. This disparity was expected given the more recent introduction of the ASD diagnosis, and the more enduring symptom profile associated with PTSD. Given the considerable amount of literature pertaining to PTSD, selective examples of issues relevant to this series of studies are presented, and existing literature reviews are referenced for further information. The theoretical framework of the empirical studies, and the rationale for the selection of the specific independent and dependent variables, are presented in chapters two to four.

Chapter two presents the historical evolution of the diagnostic classification of posttraumatic psychological responses, and describes the relevant DSM-IV (APA, 1994) diagnostic entities. The chapter illustrates that, in addition to the formal posttraumatic diagnoses of PTSD and ASD, other psychopathology has been associated with posttraumatic responses, including depression (e.g., Ballenger et al., 2000; Brady, Killeen, Brewerton, & Lucerini, 2000; Cascardi, O'Leary, & Schlee, 1999; Lifton, 1983; Reynolds & Brewin, 1998; Shalev, 2000; Shalev, Freedman, et al., 1998), somatoform disorders (e.g., Deering, Glover, Ready, Eddleman, & Alarcon, 1996; Elderkin-Thompson, Silver, & Waitzkin, 1998; Nijenhuis, Spinhoven, van Dyck, van der Hart, & Vanderlinden, 1998), other

anxiety disorders (e.g., Boudreaux et al., 1998; Brady et al., 2000; Deering et al., 1996), substance related disorders (e.g., Blanchard, Hickling, Barton, & Taylor, 1996; Brady et al., 2000; Brady, Sonne, & Roberts, 1995; Coffey, Dansky, Falsetti, Saladin, & Brady, 1998; Deering et al., 1996; Inaba, 1998; Najavits, Weiss, Shaw, & Muenz, 1998), personality disorders (e.g., Deering et al., 1996; Everly, 1993; Quinn, 1997), brief psychotic responses (e.g., APA, 1994; Deering et al., 1996; Hryuniak & Rosse, 1989) and dissociative disorders (e.g., Bremner, 1999a; Nijenhuis, Vanderlinden, & Spinhoven, 1998; Saxe, van der Kolk, Berkowitz, & Chinman, 1993).

It has been reported that it is not uncommon for individuals diagnosed with PTSD to also meet the criteria for one or more other DSM-IV Axis I disorders (e.g., Bloom, 1999; Deering et al., 1996; O'Brien, 1998). This severe deterioration in mental health is evidence of the life changing and debilitating effects of trauma exposure for some individuals. Chapter two highlights that trauma exposure may also result in psychological benefits (e.g., Waysman, Schwarzwald, & Solomon, 2001), a concept relevant in later chapters regarding adaptive psychological recovery.

The review of the aetiological models of posttraumatic stress responses in chapter three concludes that complex, multivariable processes are involved in the development of diagnostically distinct disorders. Chapter three serves as a reference point for the interpretation of the empirical findings, and proposes an integrated aetiological model of the differential development of PTSD, ASD and subclinical responses that provides the theoretical framework for the series of studies. In chapter four, multimodal assessments are advocated to comprehensively examine the variables that mediate posttraumatic psychological outcomes, a

proposal supported by previous literature (e.g., Hickling, Taylor, Blanchard, & Devineni, 1999; Keane, Wolfe, & Taylor, 1987; Quinn, 1997).

The four empirical studies utilize multimodal assessment tools to comprehensively examine a range of biopsychosocial variables associated with PTSD, ASD and subclinical responses to MVA trauma. An examination of the nature and prevalence of MVA exposure in an Australian university student sample precedes the three studies that focus on the PTSD, ASD and subclinical experimental groups. The first study explores the nature and prevalence of MVA exposure, posttraumatic symptoms reported by the sample, and sex differences in these variables, in order to establish the appropriateness of targeting this population for participation in the subsequent studies.

The second study uses psychometric data to investigate coping strategies and belief systems associated with the development of PTSD, ASD and subclinical responses to MVA trauma. The study concludes that vulnerability to the development of PTSD is associated with greater use of maladaptive coping strategies, when compared with the coping profiles of the ASD and subclinical groups. In addition, belief systems are concluded to have no significant association with the differential development of posttraumatic stress responses in this sample.

In order to investigate other variables that may mediate psychological adjustment, a selection of responses to recall of traumatic memories are examined in the third study. The study focuses on psychophysiological and psychological responses proposed to differentiate PTSD, ASD and subclinical groups on the basis of reactivity to trauma-related and neutral stimuli. A four stage guided imagery methodology is used to compare responses to idiosyncratic cues presented in the auditory mode. The results indicate distinct patterns of psychophysiological

and psychological response for each of the three groups. The findings demonstrate that recall of the MVA itself is more psychophysiologically arousing for all groups than recall of events occurring at the MVA scene. Conversely, the findings demonstrate that recall of the events occurring at the accident scene provokes greater subjective ratings of negative emotions than recall of the MVA. Results also confirm that the MVAs are perceived as traumatic to the same degree by all groups, and that the three groups are not distinguishable in terms of experiencing peritraumatic fear of death or injury. Thus, despite experiencing similarly distressing events, the three groups embark on diagnostically distinct posttraumatic symptom courses. The results are discussed in terms of the potential utility of a four stage guided imagery methodology in the diagnosis, assessment and treatment of posttraumatic responses.

The final study explores posttraumatic outcomes associated with PTSD, ASD and subclinical responses to MVA trauma. The findings demonstrate that the three groups are distinguishable on multiple psychopathological, biopsychosocial and quality of life outcome variables. The results suggest that despite the initial disorder, the ASD group profile reflects long term adaptive recovery, and allows the identification of variables that may play a role in the adaptation.

A summary of findings of the empirical studies and discussion of their implications are presented in the final chapter. Critical analyses of the investigation and directions for future research are also presented. The present investigation provides distinct multi-variable profiles of PTSD, ASD and subclinical responses to MVA trauma based on a multimodal assessment methodology. The results support the proposition that ASD, without progression to PTSD, is a diagnostic entity that is predictive of a positive prognosis characterized by posttraumatic psychological

adjustment, despite initial distress. The findings also determine that regardless of posttraumatic diagnosis, exposure to traumatic MVAs is associated with cognitive, affective, behavioural and biological effects. The next chapter commences the presentation of theoretical background issues relevant to the interpretation of the empirical studies.

CHAPTER TWO

DIAGNOSTIC CLASSIFICATION OF POSTTRAUMATIC RESPONSES

2.1 Posttraumatic diagnoses: A historical perspective

The diagnostic classification of posttraumatic responses has progressed from anecdotal observations of the effects of traumatic events on the thoughts, behaviours and physical functioning of exposed individuals, to the current status of formally recognized diagnostic entities (e.g., Wilson, 1995). The DSM-IV (APA, 1994) and the tenth edition of the International Classification of Disorders [ICD-10] (World Health Organization, 1992) may be considered to be the current leading international systems for diagnostic classification (e.g., Brett, 1996). Given that the ICD-10 classifications of trauma responses have been described to be more related to combat trauma than civilian trauma (e.g., McFarlane, 1999), the DSM-IV system was used in the present investigation. It is acknowledged that a text revision of the DSM-IV has been published in recent years (APA, 2000), but the diagnostic criteria for PTSD and ASD remain unchanged. This chapter introduces the current state of the diagnosis of posttraumatic stress disorders, and presents a historical overview of the development of these diagnostic classifications within the DSM classification system.

Historical accounts of posttraumatic symptoms documented as early as the seventh century AD, described cognitive, behavioural and psychophysiological symptoms experienced by individuals exposed to traumatic events including combat, natural disaster, accidents and interpersonal violence. For example, O'Brien (p.7, 1998) recounted the seventh century legend of "...a wild man who went away to live alone in the woods as he was affected by the sounds and sights of terrible battle. He avoided people and lived as a hermit for several years." This account was compared with PTSD, in that the man appeared to have suffered from

the negative psychological impact of combat exposure, resulting in social withdrawal and isolation. Although the posttraumatic diagnoses of PTSD and ASD were not defined until the late twentieth century, records have suggested that posttraumatic psychological symptoms existed long before they were formally classified. Table 1 summarizes other examples of historical accounts of posttraumatic symptoms prior to the first formal diagnosis in DSM-I (APA, 1952).

Table 1.

Examples of historical accounts of posttraumatic symptoms cited in secondary sources (Daly, 1983; Everly & Lating, 1995; O'Brien, 1998; Saigh & Bremner, 1999; van der Kolk, Weisaeth, & van der Hart, 1996).

Author (date)	Trauma	Symptoms (self-report/observed)
Pepys (1666)	Fire	Sleeplessness, night terrors
Da Costa (1871)	War	Irritability and physiological disturbance
Kraepelin (1896)	Accident	Severe emotional upheaval, fear and anxiety
Freud (1917)	War	Psychological disturbances
Mott (1917)	War	Disorientation in time and place
Kardiner (1941)	War	Constriction of personality functioning
Rado (1942)	War	Trembling, impotence, hyperarousal
Adler (1943)	Fire	Psychological distress
Wolf & Ripley (1947)	Torture	Blunted affect and memory impairment
Friedman (1948)	War	Sleep disorders and subjective fears

The prevalence of posttraumatic symptoms during the first half of the twentieth century in response to war-related trauma consolidated anecdotal reports of posttraumatic responses (e.g., Bremner, 1999a). The extreme violence and trauma of the series of major international wars increased the prevalence of trauma exposure and subsequent posttraumatic symptoms, and resulted in the formal diagnostic entity of a gross stress reaction, as defined in DSM-I (APA, 1952). This entity has evolved over time as the DSM classification system has been revised. Table 2 summarizes the historical progression of the formal classifications of posttraumatic responses in the DSM series, in order to place the current classifications in context.

Table 2.

The historical progression of formal classifications of posttraumatic responses in the DSM series (APA, 1952, 1968, 1980, 1987, 1994).

DSM edition	Posttraumatic response classification
DSM-I (1952)	**Gross stress reaction** "Exposure to severe physical demands or extreme stress, such as in combat or civilian catastrophe." (p.40).
DSM-II (1968)	**Transient situational disturbance** "Transient disorders of any severity (including those of psychotic proportions) that occur in individuals without any underlying mental disorders and that represent an acute reaction to overwhelming emotional stress." (p.48).

(Table continued...)

Table 2 (continued...)

DSM edition	Posttraumatic response classification
DSM-III (1980)	**Posttraumatic Stress Disorder**
	"The development of characteristic symptoms following a psychiatrically traumatic event that is generally beyond the realm of normal human experience." (p.236).
	Four defined symptom clusters
DSM-III-R (1987)	**Posttraumatic Stress Disorder**
	Modification of four symptom clusters
	More detailed information regarding age-specific features
DSM-IV (1994)	**Posttraumatic Stress Disorder**
	Amended definition of traumatic event
	Modification of four symptom clusters
	More detailed information regarding age-specific features
	Acute Stress Disorder
	New diagnostic entity

Undoubtedly, the diagnostic classification of posttraumatic responses will continue to be subject to revision as the field of traumatology continues to expand knowledge of the development and maintenance of psychiatric responses to trauma (e.g., Anthony, Lonigan, & Hecht, 1999). The current state of posttraumatic psychological diagnosis will now be reviewed.

2.2 Current diagnostic entities

The current conceptualization of posttraumatic responses as psychiatric entities has been formally recognized by the inclusion of PTSD and ASD in the DSM-IV (APA, 1994). As previously noted, the inclusion of ASD in the DSM-IV (APA, 1994) highlighted that posttraumatic stress may manifest in more than one presentation. Posttraumatic stress responses vary between individuals, symptoms fluctuate over time, and diagnosis is not an 'all-or-none' dichotomy of symptom presentation (e.g., Aldwin et al., 1994; Alexander, 1999; Blanchard & Hickling, 1999; Brett, 1996; Deering et al., 1996; Dougall et al., 2001; Green, Lindy, & Grace, 1985; Hickling & Blanchard, 1999; O'Brien, 1998). The current conceptualizations of posttraumatic responses have defined that the diagnoses of PTSD and ASD are applicable to all trauma types, despite it being suggested that different types of trauma result in specific symptoms, such as "Vietnam Syndrome" and "Post-Rape Syndrome" (e.g., Bremner, 1999a; Foa & Hearst-Ikeda, 1996).

2.2.1 PTSD

PTSD was introduced as a diagnostic entity in the third edition of the DSM in 1980 [DSM-III] (APA, 1980). The inclusion of this diagnosis in the anxiety disorders section of the DSM-III formally recognized that exposure to a traumatic event could result in the development of a specific and consistent range of psychiatric symptoms. Although PTSD was classified as an anxiety disorder, it also has been identified as having features of mood, dissociative and personality disorders, as acknowledged by Yehuda, Marshall, and Giller (1998). Previous references to psychiatric responses to trauma, such as the "gross stress reaction"

published in DSM-I (APA, 1952), had not detailed distinct symptom clusters (e.g., Everly & Lating, 1995). The diagnosis of PTSD has continued as a psychiatric entity with various criteria revisions in DSM-III-R (APA, 1987) and DSM-IV (APA, 1994).

By definition, PTSD has been classified to result exclusively from exposure to a traumatic event, and has been defined (APA, 1994) in terms of three major symptom clusters: reexperiencing, avoidance and numbing, and increased arousal. The complete DSM-IV (1994) diagnostic criteria for PTSD are displayed in Appendix A-1. Reexperiencing symptoms have been defined as including features such as recurrent and intrusive recollections or dreams about the traumatic event, acting or feeling as if the event were recurring, and reacting to internal or external cues that trigger recollections of the event. Avoidance and numbing symptoms have been defined as including manifestations such as efforts to avoid thinking, feeling or talking about the event; avoiding activities, places or people that arouse recollections of the event; feeling detached from others; and an inability to remember important aspects of the event. Increased physiological arousal has been defined as including sleeping difficulties, mood swings, concentration difficulties, hypervigilance and exaggerated startle responses. PTSD may be diagnosed only after the full constellation of symptoms has been present for more than one month following trauma. These symptoms must cause clinically significant distress or impairment in social, occupational, or other important areas of functioning in order for a diagnosis of PTSD to be applicable (APA, 1994).

The DSM-IV (APA, 1994) criteria for a traumatic event have specified that witnessing an event, without direct personal involvement, may result in the development of posttraumatic symptoms. In the current age of audiovisual and

communications technology, traumatic events are internationally exposed in detail. For example, the images of the terrorist attacks on the twin towers of the World Trade Center in the USA on September 11, 2001, were seen around the world via multimedia. Witnessing the images of these attacks may have resulted in posttraumatic responses in many individuals, including those far removed from the scene and victims, as demonstrated by previous examinations of the relationship between exposure to traumatic material via multimedia and posttraumatic stress symptoms (e.g., Klingman, 1994; Morland, 2000; Pfefferbaum et al., 2000; Veraldi & Veraldi, 2000). Clearly, most individuals are exposed to potentially traumatic events, either directly or indirectly, during their lifetime.

PTSD has been reported to affect up to 14% of the population of the USA at some point during the life-span (e.g., Sherman, 1998; Yehuda et al., 1998), 2% of the population of the United Kingdom (Jenkins & Meltzer, 1995), and 1% of the population of Australia (Creamer, Burgess, & McFarlane, 2001) with prevalence estimates varying based on detection methods. Estimates of the prevalence of PTSD among individuals exposed to a traumatic event meeting DSM-IV criteria (APA, 1994) have been reported to range from 3 to 58%, reflecting that some traumatic events were more likely to result in PTSD than others, and that the prevalence of PTSD differed between populations and events (e.g., Yehuda et al., 1998). PTSD has been found to be prevalent in approximately 25% of traumatic MVA survivors (e.g., Ehlers, Mayou, & Bryant, 1998). Sex differences have been reported in trauma exposure and PTSD prevalence, with males reporting higher rates of exposure to traumatic events, and females experiencing higher rates of PTSD (e.g., Keane, 1998; Ursano et al., 1999).

2.2.2 ASD

The inclusion of ASD in the anxiety disorders section of the DSM-IV was the first formal recognition that exposure to a traumatic event may result in the development of an acute psychiatric disorder, in addition to the more chronic disorder of PTSD (e.g., Koopman, 2000; Marshall, Spitzer, & Liebowitz, 1999). The complete diagnostic criteria for ASD are displayed in Appendix A-2. The introduction of the diagnosis of ASD emphasized the importance of dissociative symptoms in the constellation of posttraumatic psychological responses (e.g., Spiegel, Koopman, Cardena, & Classen, 1996; Spiegel, Koopman, & Classen, 1994; van der Kolk, McFarlane, & Weisath, 1996).

The ASD diagnosis has been defined as a short term constellation of symptoms, lasting between two days and four weeks posttrauma, including dissociative phenomena, reexperiencing symptoms, increased arousal and avoidance behaviour (APA, 1994). By definition, the diagnostic criteria for PTSD and ASD have considerable overlap in symptoms. The four main differences between the classifications of PTSD and ASD have been identified to be symptom duration, the specific emphasis on dissociative phenomena in the ASD criteria, the number of symptoms required within each cluster to meet diagnostic criteria, and the level of impairment resulting from symptoms (e.g., Hickling & Blanchard, 1999; O'Brien, 1998). ASD has been reported to be prevalent in approximately 13% of MVA survivors (Bryant & Harvey, 2000a).

ASD has been considered to be predictive of the development of PTSD (e.g., Birmes et al., 2001; Brewin, Andrews, Rose, & Kirk, 1999; Classen, Koopman, Hales, & Spiegel, 1998; Harvey & Bryant, 1999d; Holeva, Tarrier, & Wells, 2002; O'Brien, 1998; Winston et al., 2002). While there is some support for

this proposition, the relationship between the two diagnostic entities is likely to be complex. Despite reported rates of ASD progressing to PTSD in 63 to 83% of cases (e.g., Brewin et al., 1999; Bryant, Guthrie, Moulds, & Harvey, 2000; Bryant & Harvey, 1998; Bryant, Moulds, & Guthrie, 2000; Harvey & Bryant, 1998; Harvey & Bryant, 1999d), substantial numbers of individuals (17 to 37%) diagnosed with ASD had not developed PTSD at the time of assessment. In an Australian study of workers exposed to an industrial accident, Creamer and Manning (1998) found that 6% of the sample developed ASD, and they did not develop PTSD. Thus, the fact that ASD is predictive of PTSD in all cases has not been effectively established (e.g., Bryant, 2000; Marshall et al., 1999).

It is unclear from the research to date how many of the individuals diagnosed with ASD received treatment for their symptoms during the acute symptom phase. A study investigating the effectiveness of cognitive-behaviour therapy for ASD (Bryant, Harvey, Dang, Sackville, & Basten, 1998) found that only 17% of the ASD group who received treatment were found to have PTSD at the six month follow-up. This study suggests that early intervention may prevent PTSD. The findings may also suggest that PTSD does not always develop after ASD regardless of intervention provided. As concluded by Yehuda and Wong (2000), ASD has features that are not directly associated with PTSD, and ASD does not always lead to the development of PTSD. Bryant (2000) stated that the proportion of individuals exposed to trauma who develop PTSD are the minority of trauma survivors, regardless of higher rates of initial distress including ASD.

In light of these results, it may be suggested that there are two subtypes of ASD, one that precedes the development of PTSD, and another that leads to early symptom resolution. As previously stated, there is comparatively little research

regarding ASD when the wealth of literature pertaining to PTSD is considered. However, it is worth acknowledging that Bryant, Harvey, and colleagues have recently published a series of investigations of ASD, and their findings will be referred to in later chapters (e.g., Bryant, Guthrie, & Moulds, 2001; Bryant & Harvey, 1999; Bryant & Harvey, 2000a; Bryant & Harvey, 2000b; Bryant, Moulds, & Guthrie, 2001; Bryant & Panasetis, 2001; Guthrie & Bryant, 2000; Harvey & Bryant, 2000a, 2000b).

Considerable debate surrounded the development of the ASD diagnosis and its subsequent inclusion in the DSM-IV (APA, 1994). The previously noted studies presented by Bryant, Harvey and colleagues are particularly comprehensive regarding this issue (e.g., Bryant, Guthrie, et al., 2001; Bryant & Harvey, 1999; Bryant & Harvey, 2000a; Bryant & Harvey, 2000b; Bryant, Moulds, et al., 2001; Bryant & Panasetis, 2001; Guthrie & Bryant, 2000; Harvey & Bryant, 2000a, 2000b). In addition, the *American Journal of Psychiatry* has published a series of critical reviews from various authors during recent years focusing on the current ASD diagnosis (e.g., Butler, 2000; Koopman, 2000; Marshall et al., 1999; Simeon & Guralnik, 2000). There are several reported reasons as to why ASD was constructed as a diagnostic entity. These include the recognition of a need to identify individuals at risk of developing PTSD and to facilitate early intervention, and to identify the role of dissociative symptoms in trauma responses (e.g., Bryant, 2000; Yehuda & Wong, 2000). However, the introduction of the entity has been criticized due to limited empirical justification when compared with other DSM-IV diagnoses (APA, 1994), the view that the entity may pathologize adaptive processes occurring during the acute posttrauma phase, and the inclusion of dissociative symptoms that is not consistent with the PTSD diagnosis (e.g., Bryant,

1999; Bryant, 2000). It has been argued that future revisions of the ASD criteria need to be based on comprehensive empirical evidence (e.g., Bryant & Harvey, 2000a). It may be deduced from the considerable debate regarding the ASD diagnosis that there is not a universal school of thought regarding the entity and its relevance within theoretical and practical frameworks. It is anticipated that this research may contribute to discussion regarding the conceptual basis of the diagnosis of ASD.

It is proposed that research that investigates individuals with a diagnosis of ASD without progression to PTSD is the key to understanding the ASD entity, as it is this posttraumatic course that may provide valuable information about prevention of the development of PTSD. Regardless of the original intentions of the development of this diagnosis as a formal entity, it may have utility in understanding positive, rather than negative, posttraumatic prognoses. For example, individuals with ASD who do not progress to develop PTSD may have characteristics or be exposed to influences that protect them from developing PTSD. If the ASD diagnosis is evaluated in isolation from ASD that progresses to PTSD, then it is proposed that factors that prevent the development of PTSD, or ameliorate posttraumatic symptom severity, may be identified and potentially lead to the facilitation of early intervention and prevention treatment strategies.

2.3 Subclinical responses

Posttraumatic symptoms that do not meet the criteria for PTSD have been given various labels such as sub-syndromal PTSD, partial PTSD, sub-threshold PTSD and subclinical responses (e.g., Andrykowski & Cordova, 1998; Blanchard

& Hickling, 1997; O'Brien, 1998; Stein, Walker, Hazen, & Forde, 1997; van der Kolk et al., 1996). For descriptive purposes in the present investigation, responses that did not meet the diagnostic criteria for PTSD or ASD were termed subclinical responses. It should be clarified that this was a heterogeneous group in terms of posttraumatic psychological responses. That is, the group included individuals who exhibited a range of posttraumatic and comorbid symptoms which did not meet the criteria for PTSD or ASD. Individuals that did not report experiencing any posttraumatic symptoms were not included in this group.

Although it may be argued that this category is too broad to be of clinical value, it is proposed that the current diagnostic classifications provide no provision for the classification of posttraumatic symptoms that do not meet the criteria for PTSD or ASD. Therefore, it was deemed necessary to investigate this heterogeneous group in order to comprehensively evaluate the nature of posttraumatic responses currently outside of the realms of formal diagnostic classification. It may be that following investigation of this group, an evaluation of the inclusiveness of the existing diagnostic categories may be possible, and recommendations made regarding the clinical and research utility of assessment of all posttraumatic psychological sequelae causing distress, and those indicative of adjustment.

2.4 Comorbidity

In addition to the diagnoses of PTSD and ASD, the comorbid development of other psychiatric diagnoses following trauma exposure has been reported. Psychopathology profiles of PTSD have included high rates, in excess of 75%, of comorbidity with depression, somatoform disorders, other anxiety disorders,

substance-related disorders, personality disorders, dissociative disorders, and brief psychoses, (e.g., Blanchard, Buckley, Hickling, Taylor, 1998; Blanchard, Hickling, Taylor, & Loos, 1995; Bremner, 1999a; Breslau, Davis, Andreski, & Peterson, 1991; Bryant, 1998; Deering et al., 1996; Everly, 1993; Garfield, Fichtner, Leveroni, & Mahableshwarkar, 2001; Gerrity, Keane, & Tuma, 2001; Koch & Taylor, 1995; O'Brien, 1998; Orsillo, Roemer, Litz, Ehlich, & Friedman, 1998; Roemer, Litz, Orsillo, Ehlich, & Friedman, 1998; Shalev, 2000; Tryon, 1998). PTSD comorbidity has been reported to be complicated by the temporal sequence of onset and interactions of comorbid disorders (e.g., Frances, 1997). It has been suggested that patterns of comorbidity may differ when different trauma types are considered, and that psychiatric disorders associated with PTSD are not truly comorbid, but interwoven with the symptoms of PTSD (e.g., Deering et al., 1996). It has been reported that the constellation of PTSD symptomatology is diffuse, and that individuals diagnosed with PTSD tend to endorse a broad range of symptoms (e.g., Davidson & Foa, 1991; Keane & Wolfe, 1990).

Diagnoses commonly reported to be comorbid with posttraumatic stress disorders will now be presented, in order to describe the framework in which PTSD and ASD develop. It is noted that temporal comorbidity with ASD is not possible in most cases, due to the limited duration criteria of ASD (e.g., Bryant & Harvey, 2000a).

2.4.1 Depression

The diagnosis of Major Depressive Disorder is the most common form of posttraumatic comorbid psychopathology (e.g., Ballenger et al., 2000; Blanchard, Hickling, Taylor, & Loos, 1994a; Brady et al., 2000; Cascardi et al., 1999;

Constans, Lenhoff, & McCarthy, 1997; Deering et al., 1996; Engdahl, Speed, Eberly, & Schwartz, 1991; Green et al., 1992; Hryuniak & Rosse, 1989; Kuch, Cox & Evans, 1996; Quinn, 1997; Reynolds & Brewin, 1998; Shalev, 2000; Shalev, Freedman, et al., 1998). The United States National Comorbidity Study (Kessler et al., 1995) found that 48% of individuals diagnosed with PTSD had comorbid Major Depressive Disorder, and 22% had comorbid Dysthymia. In a sample of MVA-exposed individuals (N = 158), 35% were diagnosed with PTSD, and 53% of the PTSD group also met the diagnostic criteria for comorbid major depression (Blanchard, Hickling, Taylor, et al., 1995). Buckley (2000) reported that between 3 and 53% of treatment seeking MVA survivors with PTSD had a comorbid mood disorder such as Major Depressive Disorder.

There is some overlap in the DSM-IV (APA, 1994) diagnostic criteria for PTSD and Major Depressive Disorder. Thus, it is not surprising that depressive symptoms have been reported to be comorbid with posttraumatic stress responses (Bremner, 1999a). For example, the development of depression following traumatic experiences has been associated with insomnia (e.g., Ford & Kamerow, 1989; Mellman, Byers, & Augenstein, 1998; Neylan et al., 2001), and the effects of sleep deprivation have been reported to overlap with PTSD symptoms, such as irritability and concentration difficulties (e.g., Newhouse et al., 1989). It has been suggested that nightmares and distressing reexperiencing of traumatic memories may impair an individual's ability to experience intact sleep during which restorative functions may take place, and adaptive emotional processing may be completed (e.g., Mellman et al., 1998).

Major Depressive Disorder has been noted as a common independent consequence of exposure to trauma, and has been identified as a predisposing risk

factor for the development of PTSD (e.g., Blanchard, Buckley, et al., 1998; Brady et al., 2000; Shalev, Freedman, et al., 1998). Another consideration with regard to depressive symptoms is that the posttraumatic responses of grief, bereavement and survivor guilt have been reported to significantly impact on posttraumatic mood and symptom presentation (e.g., Figley, Bride, & Mazza, 1997; O'Brien, 1998).

2.4.2 Somatoform disorders

Somatoform disorders have been widely reported to develop comorbid to PTSD (e.g., Deering et al., 1996; Elderkin-Thompson et al., 1998; Engdahl et al., 1991; Nijenhuis, Spinhoven, et al., 1998). By definition, somatoform disorders are characterized by somatic or physical symptom presentations that are not fully explained by a medical condition, and they are also not intentionally produced (APA, 1994). Somatic rather than psychological manifestations of posttraumatic responses have been determined to be more prevalent in certain cultures and with particular nationalities (e.g., O'Brien, 1998; Terheggen, Stroebe, & Kleber, 2001). In relation to MVAs, a review by Blanchard and Hickling (1997) of comorbidity studies highlighted that 9-29% of the MVA-exposed individuals with PTSD had comorbid somatoform disorders, with many others reporting additional pain-related problems. Individuals with somatic complaints comorbid to PTSD were more likely to present for primary health care than psychological therapy (e.g., Elderkin-Thompson et al., 1998; Hickling & Blanchard, 1992). This pattern of help-seeking behaviour, and the physical manifestation of psychological problems, may be of utility in the identification of individuals who may benefit from posttraumatic psychological treatment.

2.4.3 Anxiety disorders

Panic Disorder, Agoraphobia, Generalized Anxiety Disorder, Obsessive-Compulsive Disorder, Social Phobia and Specific Phobia have been associated with the occurrence of PTSD (e.g., Boudreaux et al., 1998; Brady et al., 2000; Deering et al., 1996; Engdahl et al., 1991; Green et al., 1992). Smith and Bryant (2000) stated that individuals with ASD or PTSD may be more susceptible to developing other anxiety disorders. Buckley (2000) reported that in a large scale study of MVA survivors seeking treatment, 27% had an anxiety disorder in addition to PTSD, and 15% reported driving phobia. Consistent with these results, Maes, Mylie, Delmeire, and Altamura (2000) found in a study of fire and MVA-exposed individuals, 21% of those participants diagnosed with PTSD were also diagnosed with Agoraphobia, and 25% with Generalized Anxiety Disorder. As PTSD and ASD are both categorized in the anxiety disorders section of the DSM-IV (APA, 1994), it is not surprising that other anxiety disorders have been widely reported to develop comorbid to posttraumatic stress disorders.

2.4.4 Substance use disorders

The high rate of substance use disorders associated with posttraumatic stress has been well documented (e.g., Blanchard, Hickling, Barton, et al., 1996; Brady et al., 2000; Brady et al., 1995; Coffey et al., 1998; Deering et al., 1996; Fullilove, Lown, & Fullilove, 1992; Hryuniak & Rosse, 1989; Inaba, 1998; McLeod et al., 2001; Najavits et al., 1998; Stewart, 1996). The comorbidity of substance use disorders with PTSD has been proposed to result from attempts to self-medicate to suppress PTSD and associated symptoms (e.g., Brady et al., 2000; Nishith, Resick, & Mueser, 2001). Alternatively, it has also been proposed that

substance use disorders and PTSD have a 'shared vulnerability' in terms of aetiology, and that substance use may be a response to posttraumatic environmental stressors in combination with genetic factors, rather than a response to experiencing PTSD symptoms (e.g., McLeod et al., 2001).

Substances that have commonly been linked with posttraumatic stress reactions are alcohol, cannabis, nicotine, and benzodiazepines (e.g., Brady et al., 2000; Coffey et al., 1998; Deering et al., 1996; Fullilove et al., 1992; Hryuniak & Rosse, 1989; Inaba, 1998; Najavits et al.,1998). It has been reported that the effects of substance use may exacerbate PTSD symptoms such as irritability, impaired social interaction, and increased arousal. Substance misuse has also been reported to sabotage professional treatment approaches to PTSD (e.g., O'Brien, 1998).

In contrast, in relation to MVA-exposed individuals, Blanchard and Hickling (1997) reported no difference in substance use disorder prevalence in diagnostically distinct subgroups, and little report of alcohol or drug related problems in the sample. These findings were considered to be due to the unavoidable recruitment bias of voluntary participants, as opposed to a true reflection of comorbidity rates.

2.4.5 Personality disorders

Personality traits have been described as predisposing factors that may affect vulnerability to the development of PTSD following exposure to a traumatic experience (e.g., Paris, 2000). In terms of outcome of traumatic exposure, posttraumatic stress has been associated with the development of personality disorders such as Borderline Personality Disorder (e.g., Allen, 2001; Deering et al.,

1996; Everly, 1993; Quinn, 1997). This association has been predominantly based on exposure to sexual trauma (e.g., Kozel, 2001). Given that personality disorders are associated with enduring characteristics (e.g., APA, 1994), the development of a personality disorder following trauma exposure may be considered a severe psychiatric consequence. It should also be acknowledged that there are difficulties associated with determining pretrauma versus posttrauma personality development, and that a traumatic experience may exacerbate the symptoms of a pre-existing personality disorder that may predispose the individual to the development of PTSD (e.g., Blank, 1993).

Bollinger, Riggs, Blake and Ruzek (2000) conducted an investigation of the prevalence of personality disorders among combat veterans with PTSD (n = 107), and found that 79.4% were diagnosed with at least one personality disorder. Of this sample, 29.9% received one diagnosis, 21.5% had two diagnoses, 15.9% had three diagnoses, and 12.1% had four or more personality disorder diagnoses. Avoidant, Paranoid, Obsessive-Compulsive, and Antisocial Personality Disorders were reported as the most frequent diagnoses (47.2, 46.2, 28.3, and 15.1% respectively). As this study used an inpatient population many years following trauma exposure, it is not possible to directly attribute the development of these disorders to the combat experience. Koenen (1999) did examine whether antisocial traits were a vulnerability for, or consequence of, combat related PTSD. The findings indicated that while antisocial behaviours in childhood were associated with the later development of PTSD following combat exposure, antisocial behaviours in adulthood were identified as both predisposing and consequential to combat trauma exposure.

In terms of comorbidity of personality disorders and posttraumatic responses in MVA survivors, Blanchard and Hickling (1997) reported that 13.3% of the sample (N = 158) were diagnosed with an axis II personality disorder (APA, 1994). There were no significant differences in personality disorder prevalence between the three diagnostic groups (PTSD, sub-syndromal PTSD and non-PTSD). Sub-syndromal PTSD was defined as a posttraumatic response characterized by meeting criterion B (reexperiencing) and either C (avoidance and numbing) or D (hyperarousal), but not both. The non-PTSD group included individuals meeting the criteria for a maximum of one criterion. It was noted that within the PTSD group, the most common comorbid personality disorder was the obsessive-compulsive type. In addition, Ursano et al. (1999) reported that having an axis II personality disorder increased the risk for developing PTSD, and proposed that this may be due to the presence of a personality disorder reducing the influence of positive factors such as social support.

2.4.6 Dissociative disorders

Psychological trauma has been widely reported to trigger dissociative states (e.g., Bremner, 1999b; Nijenhuis, Vanderlinden, et al., 1998; Sandberg, Lynn & Matorin, 2001; Saxe et al., 1993; Simeon, Guralnik, & Schmeidler, 2001), as exemplified by the dissociative symptom cluster in ASD (APA, 1994). Trauma has also been linked with the development of Dissociative Identity Disorder, formerly termed Multiple Personality Disorder (e.g., Branscomb, 1991; Brende, 1987; McDowell, Levin, & Nunes, 1999; Spiegel, 1991). Dissociative states are sudden alterations in behavior, affect, sensation, perception, and knowledge, including states of dissociative fugue and amnesia (APA, 1994). Dissociation is a process

whereby information is not stored, associated or retrieved in the usual way, and information is perceived to be separated from consciousness (e.g., van der Kolk, 1999). These states have been considered to serve a defensive function (e.g., Nijenhuis, Spinhoven, et al., 1998), and have been reported to be a behavioural manifestation of stress-induced changes in the central nervous system (e.g., Bremner, Southwick, & Charney, 1999), with both psychological and physiological components. Dissociation has been said to occur along a continuum from minor dissociations in everyday life such as daydreaming, through to more severe forms such as dissociative amnesia in response to trauma (e.g., van der Kolk, 1999).

Peritraumatic dissociation has been proposed as a predictor of the development of PTSD (e.g., Harvey & Bryant, 1998; Holen, 1990; Sandberg et al., 2001; Spiegel, 1991). However, contradictory findings have suggested that peritraumatic dissociation protects against the subsequent onset of psychiatric morbidity, and that it may be a healthy, adaptive coping strategy (e.g., Lundin, 1996; Malt, Blikra, & Hoivik, 1989; Malt & Olafsen, 1992; van der Kolk, 1999). These differing opinions seem to be reconciled by the evaluation of dissociation on a continuum, whereby short term, adaptive dissociation may be viewed positively, and severe, maladaptive dissociation that negatively affects daily functioning may be considered dysfunctional (e.g., van der Kolk,1997). Suggestions for the next DSM revision have been reported to include a dissociative symptom cluster in the PTSD description, and to introduce a posttraumatic dissociative disorder diagnosis (Bremner, 1999a).

2.4.7 Brief Psychotic Disorder with Marked Stressor

By definition, exposure to a traumatic event may result in the development of a Brief Psychotic Disorder with Marked Stressor (e.g., APA, 1994; Deering et al., 1996; Famularo, Fenton, Kinscherff, & Augustyn, 1996; Hryuniak & Rosse, 1989). However, this response is considered to be one of the least prevalent comorbid diagnoses to PTSD (e.g., O'Brien, 1998), and Blanchard and Hickling (1997) reported no comorbidity of PTSD and Brief Psychotic Disorder with Marked Stressor in MVA-exposed individuals.

2.5 Posttraumatic psychological benefits and liabilities

The focus on posttraumatic diagnoses may mistakenly lead to the assumption that exposure to a traumatic event usually results in psychiatric illness. It is imperative to state clearly that trauma does not result in psychiatric illness for all people, and that the majority of individuals exposed to traumatic events recover psychologically, despite being affected by their experience (e.g., O'Brien, 1998). It is these individuals who should be studied in order to learn about the prevention of PTSD and the correlates of posttraumatic adjustment.

In addition, psychological benefits have been reported to result from traumatic experience (e.g., Aldwin et al., 1994; Fontana & Rosenheck, 1998; Tedeschi & Calhoun, 1996; Waysman et al., 2001). The proposal that posttraumatic responses may result in life changing personal growth has been supported by previous literature (e.g., Aldwin & Stokols, 1988; Lyons, 1991; McFarlane & Yehuda, 1996; Valent, 1999). Psychological benefits and liabilities have been reported to be largely independent of one another, and both described as

positively related to the severity of trauma (e.g., Aldwin et al., 1994; Fontana & Rosenheck, 1998; Sledge, Boydstun, & Rabe, 1980).

Aldwin and Stokols (1988) proposed that positive and negative consequences of events may be assessed at multiple levels of analysis, including physiological, cognitive, affective, behavioural, social and cultural levels. Positive outcomes such as increased self esteem by viewing one's actions in response to a traumatic event with pride, have been proposed to reduce the likelihood of poor psychological outcomes (e.g., Beaton, Murphy, Johnson, Pike, & Corneil, 1998).

2.6 Summary

Posttraumatic symptoms have been documented for centuries. The evolution of diagnostic classification systems, in combination with the increased prevalence trauma exposure associated with multiple international wars in the early twentieth century, led to formal diagnostic classifications of posttraumatic stress disorders. PTSD and ASD are internationally recognised entities characterized by clinical posttraumatic psychological responses. The experience of posttraumatic symptoms that do not meet the criteria for PTSD or ASD may be termed a subclinical response. Depression, somatoform disorders, other anxiety disorders, substance-related disorders, personality disorders, dissociative disorders and brief psychoses may be comorbid to PTSD. Exposure to a traumatic event may not always result in the development of posttraumatic symptoms, and posttraumatic psychological benefits such as personal growth have been acknowledged. Individual differences in posttraumatic responses have been attributed to multiple

variables, and theoretical models of the aetiology of posttraumatic stress disorders are the focus of discussion in the next chapter.

CHAPTER THREE

AETIOLOGICAL MODELS OF POSTTRAUMATIC STRESS DISORDERS

3.1 The aetiology of posttraumatic stress disorders

Central to investigations of posttraumatic psychological responses has been the search for an explanation of why some individuals exposed to a traumatic event develop long term and debilitating psychiatric illnesses, whereas other individuals exposed to the same traumatic event may demonstrate few, if any, adverse effects. A common link between existing theoretical models of the development of posttraumatic psychopathology has been that they are largely based on cognitive, affective, behavioural and physical variables. The link between thoughts, emotions, actions and bodily responses has been the basis of many models attempting to explain the complex theoretical basis of posttraumatic clinical presentations (e.g., Creamer, 1993; McGorry, 1995; van der Kolk, Weisaeth, et al., 1996; Wilson & Keane, 1997).

Historically, posttraumatic symptoms have been considered to result from constitutional vulnerability or genetic predisposition for neurosis, as noted by Yehuda and McFarlane (1995). The evolution of the diagnostic criteria for clinical posttraumatic responses has shifted the recognised aetiology of such disorders from pre-morbid characteristics and vulnerability, to the nature and intensity of the trauma (e.g., APA, 1994; McGorry, 1995). It may be argued that both genetic and environmental variables mediate posttraumatic responses (e.g., McLeod et al., 2001), as it may be considered difficult to separate the effects of both influences in order to determine if one dominates the other. For example, it has been reported that individuals exposed to trauma who have parents with PTSD are more likely to develop PTSD themselves (e.g., Pelcovitz et al., 1998). It is not clear whether such

findings reflected predominantly genetic and/or environmental factors in the aetiology of PTSD.

Predisposing, peritraumatic and posttraumatic variables have been attributed with mediating the nature and course of posttraumatic symptoms (e.g., Thompson, Norris, & Ruback, 1998). Given the considerable number of models proposed in the literature since the conceptualization of PTSD, it was not within the scope of the present investigation to review all models, therefore, examples of common theoretical perspectives have been selected for presentation. Comprehensive reviews of the aetiology of posttraumatic stress disorders have been previously presented (see McIvor, Van Velsen, Lee, & Turner, 1997; O'Brien, 1998; Rasmusson & Charney, 1997; Saigh & Bremner, 1999; Shalev, 1997; van der Kolk et al., 1996a). The majority of theories have referred to the development of PTSD without consideration of the aetiology of ASD, which may have reflected the more recent introduction of the ASD diagnosis, or the theoretical focus being placed on the aetiology of the more enduring disorder. Where reference has been made to ASD in the literature, those points have been raised in the review. This chapter presents examples of theoretical models proposed to explain the development of differential posttraumatic responses, and evaluates the merit and contribution of each type of model to an integrated theory.

3.1.1 Psychodynamic theories

Psychodynamic theories were popularized by Sigmund Freud in the early twentieth century (e.g., Freud, 1917). Psychodynamic theories of PTSD aetiology primarily have focused on faults within defence mechanisms and normal coping strategies being overwhelmed (e.g., Brett, 1993; Marshall, Yehuda, & Bone, 2000;

McGuire, 1990; Yehuda et al., 1998). For example, it has been said that intrusive symptoms are caused by the failure of defence mechanisms, and avoidance symptoms result from defensive over-control (e.g., Horowitz & Kaltreider, 1980).

Psychodynamic theories have focused on the reconciliation of the traumatic event and its meaning with an individual's concept of the self and the world (e.g., Calhoun & Resick, 1993; Grinberg, 1963; Horowitz, 1974; Marshall et al., 2000). They have been largely dependent on the nature of human experience, and the concept of learning through life experience. Psychodynamic theories have merit as part of an integrated aetiological theory, in that they have contributed an understanding and recognition of the influence of self-concept, individual perceptions, and learning across the life span, on posttraumatic psychological responses (e.g., Valent, 1999).

3.1.2 Learning theories

Learning, the capacity to adapt to the demands of the environment, is thought to result in changes in behaviour, language, emotions, attitudes and beliefs (e.g., McPherson, 1993). Theories of classical and operant conditioning have been proposed to explain how the stimuli associated with traumatic events can, through learning and reinforcement processes, come to elicit posttraumatic responses, and maintain maladaptive responding (e.g., Follette, Ruzek, & Abueg, 1998). A behavioural learning approach to the aetiology of PTSD has been formulated (e.g., Keane, Fairbank, et al., 1985; Keane, Zimering, & Caddell, 1985; Kolb & Multalipassi, 1982; Yehuda et al., 1998), with origins in Mowrer's (1939) two-factor theory of fear development. This approach suggested that the development of posttraumatic symptoms in humans may be parallel to the acquisition of

classically conditioned physiological and behavioural fear responses in animals (e.g., McPherson, 1993). The proposal was based on the theory that a traumatic event may act as an unconditioned aversive stimulus (UCS) that elicits extreme levels of autonomic arousal. Consequently, stimuli that were previously neutral which accompany the UCS may become conditioned stimuli capable of eliciting psychological and physical distress.

The behavioural learning approach asserted that avoidance behaviours in PTSD were the result of operant conditioning, where escape was reinforced by arousal reduction. It has been suggested that this approach is supported by the success of exposure-based therapies that primarily focus on reduction of arousal to trauma-related cues (e.g., Shalev, 1997). Fontana and Rosenheck (1998) affirmed that trauma exposure involved learning, which may change self-image, ways of relating to others, attitudes and beliefs. Therefore, learning theories of PTSD have been considered closely related to development across the life-span, with posttraumatic responses being mediated by age at exposure and other life experiences. Learning theories have merit as part of an integrated aetiological theory, in that they have contributed an understanding and recognition of the influence of cognitive and behavioural conditioning via life experience, on posttraumatic psychological responses (e.g., Shalev, 1997).

3.1.3 Developmental theories

DSM-IV (APA, 1994) classifications of posttraumatic disorders have been said to reflect multiple adjustment pathways in a developmental model (e.g., Morrissette, 1999; Sroufe, 1997). This notion provided recognition of a traumatic event as a developmental marker. That is, a traumatic event may be viewed as one

event in a lifetime, and posttraumatic antecedents, concomitants and consequences may be considered in a longitudinal developmental context. Focus has been placed on the significance of traumatic events in childhood development, particularly in terms of the development of psychopathology (e.g., Draganic, Lecic-Tosevski, & Clouska-Hertzog, 1997). As previously discussed, in adulthood trauma exposure has also been demonstrated to significantly impact on psychological development and consequential quality of life (e.g., Valent, 1999; van der Kolk, 1996b).

Developmental factors, such as cognitive, emotional and moral developmental phases, have been said to influence resilience and vulnerability to the development of psychopathology (e.g., Valent, 1999). The timing of exposure to a traumatic event in terms of an individual's developmental phase during the life-span has been considered to influence posttraumatic psychological outcomes.

The developmental pathways concept more recently presented by Sroufe (1997) may be applied to differential posttraumatic response. The concept described psychopathology as a succession of pathways leading away from competent functioning in children, and may be applied to differential posttraumatic response in adults. For example, some posttraumatic recovery pathways may exhibit little deviation from their previous course, as represented by nonpsychopathological responses to trauma. However, other pathways may be characterized by marked deviations, as represented by the development of PTSD. In line with the developmental pathways concept, the pathways may continue to diverge and/or converge over time. Divergence may be exemplified by an initial nonpsychopathological response with a delayed PTSD response. Convergence may be exemplified by an initial ASD response which later converges with the pathway of nonpsychopathological adaptive recovery. This model facilitated consideration

of trauma exposure as one event in the course of a lifetime pathway, with the antecedents, concomitants and consequences of the event occurring in a broader developmental context.

When considering the temporal sequence of life events, and the developmental stage at which trauma exposure occurs, it has been proposed that it is important to not only consider the individual, but also the socio-cultural environment in which the individual is attempting to function during and following trauma exposure (e.g., Brennan, 1998).

Developmental theories have merit as part of an integrated aetiological theory, in that they have contributed a recognition of traumatic events as developmental markers and provided a longitudinal view of psychological posttraumatic adjustment pathways (e.g., Valent, 1999).

3.1.4 Psychosocial and environmental theories

The influence of social environments and cultural beliefs on the development of posttraumatic responses has been acknowledged (e.g., Kirmayer, 1996; Valent, 1999). Psychosocial and environmental theories of the aetiology of posttraumatic responses have indicated that the nature of intimate relationships, family support, community support, and religious and cultural influences may shape, and to some extent determine, an individual's response to a traumatic event (e.g., Beckham, Feldman, et al., 1998; Brennan, 1998; Wilson & Moran, 1998).

One historical, large scale evaluation of this theory has been described using the experiences of soldiers returning from the Vietnam war. The war was reported to have resulted in social and cultural ramifications for returning soldiers, particularly evident when they attempted to reintegrate into the community after

their service (e.g., Beckham, Feldman, et al., 1998; Brennan, 1998). Displacement, isolation, political opposition to their actions, and community hostility were reportedly experienced by many soldiers, in stark contrast with the warm "hero's welcome" and appreciation expressed by communities to defence forces in previous conflicts. These socio-cultural factors were reportedly associated with the development of PTSD (e.g., Everly & Lating, 1995). In terms of civilian trauma, particularly those involving facing the fear of death, the impact of such experiences has been reported to influence religiosity and spiritual change, and conversely, predisposing religious and spiritual beliefs have been proposed to affect posttraumatic outcomes (e.g., Valent, 1999). Psychological effects in this regard have been reported as both negative and positive, with some individuals losing faith in their religion following the event, and others becoming increasingly religious in the aftermath.

Psychosocial and environmental theories have ranged from emphasizing the importance of supportive relationships and maintaining social networks to aid posttraumatic psychological recovery, to proposing the larger scale influences of community expectations and national cultures (e.g., Andrykowski & Cordova, 1998; Blank, 1993; Hayakawa, Fischbeck, & Fischoff, 2000). Psychosocial and environmental theories have merit as part of an integrated aetiological theory, in that they have contributed an understanding and recognition of the influence of societal expectations, religion, culture and social support, on posttraumatic psychological responses (e.g., Shalev, 1997).

3.1.5 Personality theories

The influences of many of the variables already mentioned, such as life experience, societal expectations, personal beliefs, and the need for social support may be mediated by personality. Personality has been defined in previous traumatology literature as a constellation of attributes that describe, explain and predict an individual's behaviour (e.g., Schnurr & Vielner, 1999). As described in the previous chapter, predisposing personality may influence posttraumatic symptom outcomes, but personality may also be affected by trauma exposure, and enduring personality changes may result. The hypothesis that posttraumatic personality profiles differ between individuals diagnosed with PTSD and individuals who do not develop the disorder has been widely supported (e.g., Bunce, Larsen, & Peterson, 1995; Keane, Malloy, & Fairbank, 1984; Schnurr & Vielner, 1999; Scott & Stradling, 1992). It has been recognised as logistically difficult to ascertain if these differences represent pretrauma disposition or posttraumatic outcomes (e.g., Valent, 1999).

Poor prognosis for posttraumatic psychological recovery has been associated with a range of posttraumatic personality variables including high trait anxiety, low self-esteem, high neuroticism, high introversion, emotional instability, tendency to isolate from others, emotional lability, poor anger control, paranoia, confusion, and feelings of emptiness and boredom (e.g., Bunce et al., 1995; Mihaescu & Baettig, 1996; Richmann & Frueh, 1996). In contrast, positive prognosis for posttraumatic psychological recovery has been associated with psychologically protective factors such as hardiness, self-reliance, acceptance of trauma as a challenge to be overcome, personal faith, sense of humour and ability to engage in supportive relationships (e.g., Benezra, 1996; Waysman et al., 2001).

Positive apocalypse consciousness, a personality variable described by Browne-Miller (1996), has been defined as an individual's ability to meet the challenges of traumatic experience and to see positive outcomes resulting from a catastrophe. This concept is provided as another example of a personality variable that has been proposed to mediate posttraumatic outcomes.

Personality theories have merit as part of an integrated aetiological theory, in that they have contributed an understanding and recognition of the influence of individual traits on posttraumatic psychological responses, and conversely, the role of traumatic experiences in affecting personality change (e.g., Scott & Stradling, 1992).

3.1.6 Information processing and cognitive theories

Information processing and cognitive theories of the aetiology of posttraumatic stress disorders have focused on the mechanisms by which trauma-related information is processed, and the content of the information. Variables such as executive functioning abilities, semantic networks and individual differences in environmental perception have been identified as mediating influences in the development of posttraumatic symptoms (e.g., Bryant & Harvey, 1997, 2000a; Dearden, 2000; Follette et al., 1998; Stanford, Vasterling, Mathias, Constans, & Houston, 2001).

The development of PTSD has been proposed to result from failure to adequately process trauma-related information, and maladaptive cognitive processes, increasing the severity of symptoms (e.g., Amir, Stafford, Freshman, & Foa, 1998; Bryant & Harvey, 1996; Buckley, Blanchard, & Neill, 2000; Foa & Kozak, 1991; Joseph, Williams, & Yule, 1995; Litz et al., 1996; Sachs & Peterson,

1996; Vasterling, Brailey, Constans, & Sutker, 1998). PTSD has been reported to result in a breakdown of a number of processes associated with executive functioning including attention, controlled learning, verbal fluency and memory functions (e.g., Galletly, Clark, McFarlane, & Weber, 2001; Gilbertson, Girvits, Lasko, Orr, & Pitman, 2001; van der Kolk, 1996c; van der Kolk & Fisler, 1995; van der Kolk, van der Hart, & Marmar, 1996).

There are a range of models proposed to explain the mechanisms by which these breakdowns occur. The longitudinal cognitive processing model (Creamer, Burgess, & Pattison, 1992) has been proposed to explain that cognitive processing mechanisms of posttraumatic reactions change over time, and may directly affect the manifestation of posttraumatic disorders at the symptom level. However, inconsistent findings with regard to the association between time since trauma exposure and symptom presentation have been evident in the literature. Consistent with Figley's (1978) stress evaporation hypothesis, it has been reported that posttraumatic symptoms have decreased as time since trauma exposure increased (e.g., Ursano, Fullerton, Kao, & Bhartiya, 1995). Other studies have found that posttraumatic symptoms vary over time, with active symptom phases being triggered by stressful events (e.g., Amick-McMullan, Kilpatrick, & Veronen, 1989; Creamer et al., 1992; Thompson et al., 1998).

Focusing on the actual mechanisms by which information is processed, Tryon's (1999) concept of bidirectional associative memory (BAM) provided an explanantion of PTSD based on connectionist neural network theory. This theory proposed that BAM is used to encode emotion and cognition, and that encoding is a learning process that has the potential to alter brain stuctures in ways that cannot be detected by neuroimaging techniques. Therefore, the BAM processes that may

take place in response to trauma have been proposed by Tryon (1999) to result in PTSD in some individuals. The functional links between cognitive processes and structural changes in the brain have been described, particularly in relation to memory deficits resulting from hippocampal damage secondary to increased neuroendocrine responses to conditioned stimuli in PTSD (e.g., Buckley et al., 2000).

Other examples of information processing and cognitive theories have included the cognitive content-oriented model (e.g., Follette et al., 1998) that emphasized the role of negative beliefs in causing distress and symptom maintenance; the semantic network model of internal memory structure in response to traumatization (e.g., Foa & Rothbaum, 1998), that focused on the development of fear structures in memory; the narrative model (e.g., Meichenbaum & Fong, 1993), that focused on the role of mental stories of traumatic events in the development of symptoms, rather than negative thoughts in isolation; and models of memory and emotional processing (e.g., Brewin, Dalgleish, & Joseph, 1996; McNally, 1997) that proposed the mediating role of information processing mechanisms in the development of symptoms.

Information processing and cognitive models such as these link psychological variables and underlying biological functions. The information processing and cognitive theories have not only considered posttraumatic information processing and cognitions, but have also considered peritraumatic cognitive processes to be influential in symptom development. Reportedly effective treatments of PTSD such as cognitive-behavioural, exposure and narrative therapies have been designed to target these problems, and have aimed to encourage adaptive cognitive processes, organize traumatic information, overcome

attentional and memory bias, reduce the intensity of associated emotion, and increase mastery of the material (e.g., Amir et al., 1998). Their reported success in reducing symptom severity formally verifies the validity of the contribution of information processing and cognitive variables in the aetiology of posttraumatic stress disorders.

Information processing and cognitive theories have merit as part of an integrated aetiological theory, in that they have contributed an understanding and recognition of the underlying neural networks and cognitive abilities required to process traumatic information, and the influence that these variables may have on posttraumatic psychological responses (e.g., Bryant & Harvey, 2000a).

3.1.7 Biological theories

Individuals diagnosed with PTSD have been observed to develop an enduring vigilance for, and sensitivity to, environmental threat. This sensitivity has been reported to result in altered biological responses to sensory stimuli, with the potential for generalization to innocuous stimuli (e.g., Horton, 1995; van der Kolk, 1997). Chemical alterations in the central nervous system, resulting in potentially long term functional and structural changes in the brain, have been reported to occur in response to trauma exposure (e.g., Bremner, Staib, et al., 1999; Fleming & Baum, 1987; McFall, Murburg, Roszell, & Veith, 1989; Pynoos, Steinberg, Ornitz, & Goenjian, 1997; Yehuda & McFarlane, 1997). PTSD has been associated with these types of changes in the hypothalamic-pituitary-andrenocortical axis and the sympathetic nervous system, mediated by alterations in noradrenaline, serotonin, catecholamine, and endogenous benzodiazepine and opioid systems (e.g., Bremner et al., 2000; Bremner, Southwick, et al., 1999;

Marshall & Pierce, 2000; Southwick & Friedman, 2001; Southwick, Krystal, Johnson, & Charney, 1995; Sutherland & Davidson, 1994; van der Kolk, Dreyfuss, & Michaels, 1994).

Animal studies have found that noradrenaline depletion, peripheral endorphin release, and a centrally mediated analgesic response, have all been characteristic responses to environmental shock (e.g., Krieger, 1983; Pitman, 1993; Rasmusson & Charney, 1997). For example, Nijenhuis, Vanderlinden, and colleague (1998) proposed animal defensive reactions as a model for trauma-induced dissociative reactions. They paralleled the alternating psychophysiological states associated with trauma-induced dissociative states with animal defensive and recuperative states that are evoked in response to severe threat. Dissociative theories have been proposed to explain the differential development of ASD, in that it has been suggested that individuals developing the disorder demonstrate the ability to use defensive dissociative states (e.g. Bryant & Harvey, 2000a; Krystal, Bennett, Bremner, Southwick, & Charney, 1996; Zahn, Moraga, & Ray, 1996).

To further exemplify this model, flight has been reported to be inspired by the desire to be physically removed from threat, and may be compared with human avoidance behaviours (e.g., Bolles & Fanselow, 1980; Flack, Litz, Hsieh, Kaloupek, & Keane, 2000). Freeze responses, also termed behavioural immobility, have been reported to be evoked when physical movement may reduce chances of survival. When an animal has been placed in a position of threat, freezing may have been combined with analgesia. The process has been proposed to be a functional one that allows the animal to focus on survival, and divert attention from potential physical pain, panic and fear (e.g., Vaiva, Ducrocq, Cottencin, Goudemand, & Thomas, 2000). Behaviours such as crying out for help have been reportedly

inhibited by this biological process, in order to reduce reactions that may compromise optimal defence. The response has been proposed to be mediated by endogenous opioids (e.g., Fanselow & Lester, 1988; Krystal et al., 1989; Siegfried, Frischknecht, & Nunez de Souza, 1990), and the analgesia has also been proposed to be a mechanism to reduce the pain of imminent death (e.g., Greyson, 1993). Fight responses have been reported to reflect aggressive self-defence behaviours resulting from underlying neurochemical changes (e.g., Krishnan, 1999).

When the threat has been removed, the perpetuation of these defense mechanisms has been reported to result in biological and psychological change. In humans, this may be characterized by flight reactions becoming hyperarousal and avoidant responses; freeze reactions becoming emotional numbing, anhedonia and dissociation; and fight reactions resulting in ongoing irritability and aggression (e.g., Grant-Arreola, 2000; van der Kolk, Greenberg, Boyd, & Krystal, 1985; Yehuda & McFarlane, 1995).

van der Kolk and colleagues (1985) suggested that the behavioural consequences of trauma in animals were the result of learned helplessness, and that parallel trauma responses in humans may result in catecholamine depletion and stress-induced analgesia. Biochemical theories of the aetiology of posttraumatic stress disorders have been supported by the successful use of pharmacological treatments for posttraumatic symptoms, aimed at restoring chemical balances through the modification of neurotransmitter function, including antidepressants, adrenergic agonists and antagonists, anticonvulsants, and benzodiazepines (e.g., Garfield et al., 2001; Neylan et al., 2001; Smajkic et al., 2001; van der Kolk, 2001).

The structural and functional correlates of these reported neurochemical changes have been described by various neurobiological models. For example, a neurobiological model of PTSD described by Shucard, McCabe, Szymanski, and Sparks (1998) proposed that posttraumatic neurochemical changes resulted in the dysregulation of the temporal amygdaloid complex and its interrelation with the prefrontal cortex and the locus coeruleus. This dysregulation was attributed with producing a state of hypersensitivity leading to heightened arousal in response to internal and external stimuli. Early clinical evidence for neurobiological dysfunction associated with PTSD included increased cardiovascular morbidity associated with increased autonomic reactivity (e.g., Orr, 1990); persistent hypervigilance associated with changes in adrenergic and noradrenergic functioning (e.g., McFall, Murburg, Ko, & Veith, 1990); learning and memory difficulties associated with changes in hypothalamic-pituitary-adrenal axis functioning (e.g., Pitman & Orr, 1990; Yehuda, Southwick, & Nussbaum, 1990); and chronic blunting of emotional responses associated with increased endogenous opiate functioning (e.g., van der Kolk, 1996a). Psychophysiological measures have been used to assess, at a peripheral level, these neurochemical and neurobiological theories (e.g., Blanchard, Hickling, Taylor, & Loos, 1994b; Blanchard, Hickling, Buckley, & Veazey, 1999; Blanchard, Kolb, Taylor, & Wittrock, 1989; Blanchard, Kolb, & Prins, 1991; Laor et al., 1999; Muraoka, Carlson, & Chemtob, 1998).

As previously discussed, the heightened reactivity and central nervous system changes may have resulted from any number of factors including biological vulnerability, learning effects, and cognitive appraisal. Regardless of the cause, the differences in patterns of biological response have provided an avenue for less subjective assessment in the diagnosis of specific posttraumatic stress responses,

resulting in important resources for clinical and legal practitioners. Biological theories have merit as part of an integrated aetiological theory, in that they have contributed an understanding and recognition of the physical mechanisms underlying and maintaining posttraumatic psychological responses (e.g., Shalev, 1997).

3.1.8 Integrated theories

All of the reviewed theories were considered to have merit, but no theory alone was considered to address the complexity of the aetiology of posttraumatic stress disorders. Theoretical models with integrated hypotheses of the aetiology of PTSD have been proposed, such as Everly's (1993) two-factor model of posttraumatic stress which integrated biological and psychological evidence to explain the aetiology of PTSD, and Shalev's (1997) biopsychosocial model which integrated physical, psychological and social elements to achieve the same aim.

King, King and colleagues have also published a series of studies utilizing structural equation modeling procedures that propose an integrated theory of the complex influences of multiple pretrauma, peritrauma and posttrauma variables on the severity of posttraumatic symptoms (e.g., King et al., 1996; King, King, Fairbank, Keane, & Adams, 1998; King, King, Foy, Keane, & Fairbank, 1999; King, King, Gudanowski, & Vreven, 1995; Litz, King, King, Orsillo, & Friedman, 1997). These studies found that pretrauma, peritrauma and posttrauma variables influenced symptom severity, including early trauma history, peritraumatic perceived threat, posttraumatic stressful life events, hardiness, and posttraumatic social support.

Bryant and Harvey (2000a) presented an integrated model of ASD based on network theory, with emphasis on the interaction of acute cognitive and physiological processes in symptom development and adjustment. Despite the apparently contrasting features of the aetiological models presented, all of the models share basic, complimentary constructs testable by an integrated approach (e.g., McIvor et al., 1997). Given the wide range of theories proposed, it was considered inevitable that the integration of theories would provide a more comprehensive explanation of the aetiology of posttraumatic stress disorders. An integrated model, incorporating the complex array of internal and external factors suggested by the many varied models, was proposed to provide the most holistic model of the differential development of posttraumatic stress disorders. A diagrammatic representation of the model is presented in Figure 1. The model proposes that multiple internal and external factors are influential as predisposing, peritraumatic and posttraumatic mediators of posttraumatic responses, and these influences have cognitive, affective, behavioural and physical effects that result in the differential development of PTSD, ASD, subclinical and symptom-free responses to trauma. This model is supported by other integrated approaches to the development of posttraumatic stress responses (e.g., Blanchard & Hickling, 1999; Bryant & Harvey, 2000a; Carlson, Dalenberg, Armstrong, Daniels, & Roth, 2001; Everly, 1995b).

Methodologically, it was considered difficult to test this model in a sample of individuals that may include some individuals experiencing severe posttraumatic symptoms as such a model, by definition, would require a psychologically demanding multimodal assessment. However, it was deemed necessary to choose a range of variables representing the majority of the identified areas, and to select a

battery of efficient multimodal assessment tools to test the proposed integrated aetiological model.

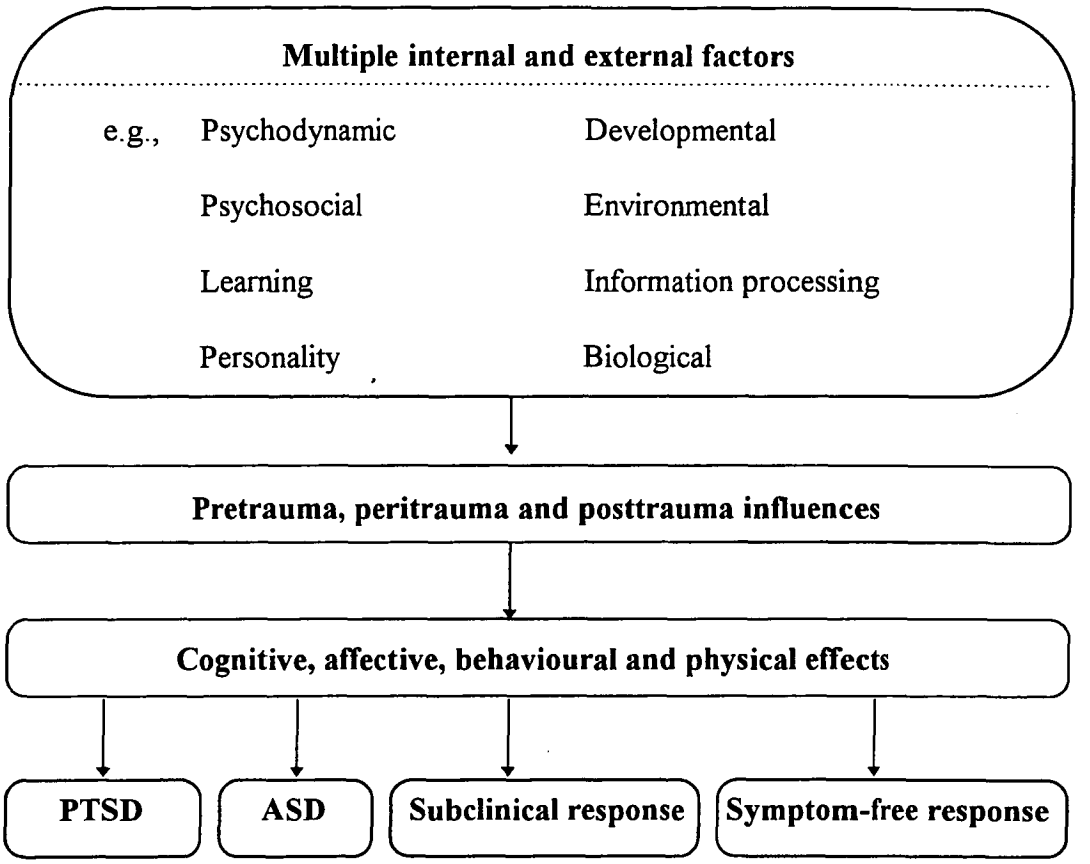

Figure 1.

An integrated model of the aetiology of posttraumatic stress disorders.

3.2 Summary

A broad range of theoretical models of the aetiology of posttraumatic stress disorders has been presented to exemplify the complexity of the differential development of posttraumatic responses, and their longitudinal course. It was acknowledged that the majority of theories referred to the development of PTSD without consideration of the aetiology of ASD, as previously presented by Bryant

and Harvey (2000a). The literature indicated that all of the aetiological models have strengths, and have contributed to the understanding and recognition of mediating variables in the differential development of posttraumatic responses. However, it was concluded that no theory alone explained the complexity of the aetiology of posttraumatic stress disorders. Common elements of the presented models were that the variables and processes that they described exerted pretrauma, peritrauma and posttrauma influences on cognitive, affective, behavioural and physical outcomes. An integrated model was proposed to represent the influence of multiple internal and external factors in the aetiology of differential posttraumatic responses. In light of the complexity of an integrated model, and the limitations of assessing a broad range of variables in a traumatized population, the benefits of selective and efficient multimodal assessment of posttraumatic responses were endorsed. In order to describe the rationale for the multimodal tools selected to achieve this task in the empirical studies, the range of tools available are outlined and discussed in the next chapter.

CHAPTER FOUR

MULTIMODAL ASSESSMENT OF POSTTRAUMATIC STRESS RESPONSES

4.1 Multimodal assessment

Comprehensive and accurate diagnostic procedures have been considered extremely important tools as they lead to appropriate diagnosis and treatment selection, and facilitate professional communication about symptom presentation and prognosis (e.g., Lating & Everly, 1995; Lating, Zeichner, & Keane, 1995; Netland, 2001; O'Brien, 1998; Weathers & Keane, 1999). These procedures may not only have implications for individual psychological recovery, but also community allocation of treatment and intervention resources, and psycholegal compensation decisions (e.g., Mayou, 1999). The complexity of the multiple theoretical explanations of the aetiology of posttraumatic stress responses, as discussed in the previous chapter, is proposed to provide the rationale for multimodal assessment of posttraumatic responses.

Some authors (e.g., Hickling, Taylor, et al., 1999; Orsillo, Batten, & Hammond, 2001; Quinn, 1997) have recommended addressing the inherent weaknesses in any one form of data collection by using multimodal assessment. Keane and colleagues (1987) emphasized the importance of multimodal assessment by presenting a multiaxial assessment procedure for PTSD in combat veterans. They proposed, on the basis of an extensive examination of existing assessment tools, that the following measures provided a comprehensive assessment: a structured clinical interview; a study of premilitary and military history; psychometric measures, including depression and anxiety inventories; and psychophysiological assessment. Kuch and colleagues (1996) concurred with the multimodal assessment approach by commenting that screening instruments and structured interviews yielded reliable assessments of PTSD following MVA

trauma. Psychometric tools, clinical interviews, and psychophysiological measures have been widely used and accepted data sources in trauma research (e.g., Gaston, Brunet, Koszycki, & Bradwejn, 1998; Lomranz, 1995; Pelcovitz et al., 1998; Suedfeld, Fell, & Krell, 1998). Keane and colleagues have been instrumental in the development and critical review of multiple assessment methods of posttraumatic symptoms (e.g., Keane, 1997; Keane, Caddell, & Taylor, 1988; Keane et al., 1998; Keane et al., 1984; Lyons, Gerardi, Wolfe, & Keane, 1988; Newman, Kaloupek, & Keane, 1996; Solomon, Keane, Newman, & Kaloupek, 1996; Wolfe, Keane, Lyons, & Gerardi, 1987).

4.1.1 Self-report measures

The logistical constraints of investigating responses to trauma have been documented (see Newman et al., 1996; Norris & Kaniasty, 1992). Given the sudden and unexpected nature of trauma, the most accessible measures have been retrospective self-report. This constraint has been documented as a weakness in some literature (e.g., Gallagher, Riggs, Byrne, & Weathers, 1998; Matorin & Lynn, 1998; Orsillo et al., 1998; Thompson et al., 1998). However, others have viewed self-report as providing information that is unavailable through other assessment mechanisms, and have expressed considerable value in the reported experience of the individual (e.g., Derogatis, 1983). Self-reported information may be formally collected by the use of structured interviews and psychometric assessment tools.

4.1.1.1 Structured interviews

Structured interviews have been designed to specifically assess PTSD and ASD (e.g., Calhoun & Resick, 1993; Carlson, 1996; Harvey & Bryant, 1999a; Orsillo, 2001). The purpose of structured diagnostic interviews is to obtain an accurate history of symptoms in order to formulate an appropriate diagnosis (e.g., Antony, Orsillo, & Roemer, 2001; McGuire, 1993). Obtaining an accurate history of the onset, nature, development and duration of symptoms is an essential element of the process (e.g., Newman et al., 1996). Antecedents and consequences of symptoms may also be used to describe mediating factors in symptom development (e.g., Orsillo, 2001). Structured interviews often have been designed to elicit information specific to research questions (e.g., Duggan & Sroufe, 1998; Thompson et al., 1998; Weathers, Ruscio, & Keane, 1999).

Examples of structured interviews for PTSD are the Diagnostic Interview Schedule [DIS] (Robins, Helzer, & Croughan, 1981), the Structured Clinical Interview for DSM-III-R [SCID] (Spitzer & Williams, 1985), and the Clinician Administered PTSD Scale [CAPS] (Blake et al., 1990). The DIS is a structured interview designed to assess a wide range of mental illnesses including PTSD, and has been adapted according to DSM revisions. The SCID is an interview that also comprehensively covers the criteria for a wide range of Axis I disorders, including PTSD (e.g., Keane, 1993). The SCID has also been adapted according to DSM revisions. The CAPS assesses both severity and frequency of PTSD symptoms using specific criteria (e.g., Orsillo, 2001). Data collection for the following series of studies commenced prior to the publication of DSM-IV (APA, 1994) compatible structured interviews. However, such tools have been published concurrent to the conduct of this research project, including the DSM-IV revisions

of the CAPS, SCID and DIS, and the Acute Stress Disorder Interview (e.g., Blake et al., 1998; Bryant & Harvey, 2000a; Newman et al., 1996; Orsillo, 2001).

4.1.1.2 Psychometric tests

Psychometric tests have been considered less intrusive, more time efficient, and relatively inexpensive when compared with other measures, such as neuroimaging and psychophysiological assessments (e.g., Solomon, Keane, et al., 1996). In addition, the format of psychometric tests, including those termed 'symptom recognition instruments' by O'Brien (1998), have been used to enable an individual to articulate the symptoms they have experienced. This process has been recognized to help overcome barriers to self-report, such as feeling ashamed of symptoms, or accepting symptoms as 'normal' (e.g., Newman et al., 1996). It has been acknowledged that a potential disadvantage of these formats is the ease of endorsement, and the problems associated with symptom suggestibility (e.g., O'Brien, 1998). These constraints have been acknowledged in the use and interpretation of psychometric measures, and measures incorporating validity control subscales have been favoured (e.g., Blanchard, Wittrock, Kolb, & Gerardi, 1988; Briere, 1995).

There have been many psychometric tests designed specifically to assess posttraumatic responses, including the Accident Fear Questionnaire (AFQ; Kuch, Cox, & Direnfeld, 1995), Davidson Trauma Scale (DTS: Davidson et al., 1997), Distressing Event Questionnaire (DEQ; Kubany, Leisen, Kaplan, & Kelly, 2000), Impact of Event Scale (IES; Horowitz, Wilner, & Alvarez, 1979; and IES-R; Weiss & Marmar, 1997), Minnesota Multiphasic Personality Inventory PTSD Scale (MMPI-PTSD; Keane et al., 1984), Mississippi Scale for PTSD (Keane et

al., 1988), Penn Inventory for PTSD (Hammarberg, 1992), PTSD Checklist (PCL; Weathers, Litz, Huska, & Keane, 1991), PTSD Symptom Scale (PSS; Foa, Riggs, Dancu, & Rothbaum, 1993), Stanford Acute Stress Reaction Questionnaire (SASRQ; Cardena, Koopman, Classen, Waelde, & Spiegel, 2000), Acute Stress Disorder Scale (ASDS; Bryant, Moulds, & Guthrie, 2000), and the Trauma Symptom Inventory (TSI; Briere, 1995). The psychometric properties and detailed descriptions of these tools may be found in a comprehensive review by Orsillo (2001).

Two trauma-specific psychometric tools were chosen for use in the present investigation. The Impact of Event Scale - Revised [IES-R] (Weiss & Marmar, 1997) was preferred due to it being a recent adaptation of the widely used IES (Horowitz et al., 1979), and its utility in the brief measurement of current symptoms experienced in response to any trauma type (e.g., Joseph, 2000). The TSI (Briere, 1995) was also selected as a more comprehensive measure of posttraumatic symptoms experienced during the six months prior to testing. The TSI has been reported to be useful in the assessment of civilian trauma, despite being developed with specific reference to sexual trauma (e.g., Briere, 1995), and incorporates consideration of dissociative symptoms relevant to the ASD diagnosis. When this project was commenced, there were no specific psychometric tools available to assess ASD. It is acknowledged that since that time, such tools have been developed and published, including the SASRQ and the ASDS (e.g., Bryant et al., 2000; Cardena et al., 2000).

Psychometric assessment instruments designed to measure other psychiatric and posttraumatic outcome variables have also been used in the assessment of PTSD, comorbidity and posttraumatic outcomes. For example, the Beck

Depression Inventory [BDI] (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), the Beck Anxiety Inventory [BAI] (Beck & Steer, 1993), the Symptom Checklist-90-Revised [SCL-90-R] (Derogatis, 1983), the Personality Assessment Inventory [PAI] (Morey, 1991), and the Quality of Life Inventory (Frisch, 1992) have been used in the evaluation of posttraumatic outcomes (e.g., Blanchard et al., 1989; Constans et al., 1997; Forbes, Phelps, & McHugh, 2001; Fullerton et al., 2000; Keane, 1993; Maker et al., 2001; McGuire, 1990; Najarian, Goenjian, Pelcovitz, Mandel, & Najarian, 2001; Nishith et al, 2001; Rothbaum et al., 2001; Sandberg et al., 2001; Scragg, Grey, Lee, Young, & Turner, 2001; Smajkic et al., 2001). These tools have also been selected for use in the present investigation due to their relevance in assessing PTSD and ASD in a larger framework of comorbidity.

4.1.2 Non self-report measures

Despite the reported advantages, the constraints of self-report measures in the assessment and diagnosis of posttraumatic responses have highlighted the need for non self-report measures as part of a multimodal assessment battery (e.g., Williston, 2001). Particularly in adversarial situations where an individual may be seeking financial compensation for psychological injury, professional evaluations of the extent of injury or disability have been advised to include non self-report measures that are reportedly less influenced by malingering or symptom exaggeration (e.g., Haines, Williams & Holmes, 2001; Rosenberg, 2001). Neuroimaging techniques and psychophysiological assessments are two non self-report methodologies via which nervous system functioning may be used to detect the presence or absence of specific types of posttraumatic responses.

4.1.2.1 Neuroimaging techniques

Specific processes underlying particular patterns of biological response may be assessed using neuroimaging techniques (e.g., Knight, 1997). Three examples of this form of assessment of posttraumatic responses are Positron Emission Tomography [PET] scanning (e.g., Bremner, Staib, et al., 1999), Single Photon Emission Computerized Tomography [SPECT] scanning (e.g., Bremner et al., 2000; Levin, Lazrove, & van der Kolk, 1999; Zubieta et al., 1999), and functional Magnetic Resonance Imaging [MRI] (e.g., Bonne et al., 2001; Bremner, 1999b; Knight, 1997; Pitman, Shin, & Rauch, 2001; Rauch et al., 2000). Studies using these techniques have contributed evidence to support the biological models of PTSD presented in the previous chapter. Not only in an assessment context, but also looking to future treatment, neuroimaging techniques in combination with guided imagery techniques and virtual reality technology have been expanding methodological possibilities (e.g., Rothbaum et al., 1999; van der Kolk, 2001). Neuroimaging techniques, by which the actual locations of activity and functions within the brain may be assessed, have been proposed to provide a comprehensive addition to the assessment battery (e.g., Knight, 1997). A current limitation of this method of assessment is access to resources and cost. However, the central nervous system studies have supported the body of knowledge produced by the peripheral psychophysiological studies, further validating the effectiveness of this more accessible method of assessment.

4.1.2.2 Psychophysiological assessment

Multimodal psychophysiological techniques have been used to assess PTSD and have been proposed as a useful tool when considering PTSD as a disorder of

arousal (e.g., Everly, 1990; Orr & Kaloupek, 1997; Pitman, 1997). Peripheral measures such as heart rate, blood pressure, skin conductance, electromyography, and finger blood volume have been used to examine patterns of psychophysiological responses (e.g., Blanchard et al., 1999; Bryant, Harvey, Gordon, & Barry, 1995; Bryant, Harvey, Guthrie, & Moulds, 2000; Orr, Lasko, et al., 1998; Shalev, Sahar, et al., 1998). The reported reliability and noninvasiveness of these measures have popularized their utility (e.g., Shalev, 1999).

PTSD, by definition, has been associated with increased psychophysiological reactivity to trauma-related stimuli (e.g., Blanchard, Hickling, Taylor, & Loos, 1994b; Blanchard et al., 1999; Blanchard et al., 1989; Blanchard, Hickling, & Taylor, 1991; Laor et al., 1999; Muraoka et al., 1998; Pitman et al., 1990; Rothschild, 2000). In a recent comprehensive review of studies that have investigated the psychophysiological assessment of PTSD, Blanchard and Buckley (1999) noted that 21 of the 31 studies involved the study of combat-related trauma, highlighting the need for investigations of civilian trauma types. The most common responses measured in the 31 studies were heart rate, electrodermal activity (skin resistance or conductance), electromyography and blood pressure.

Comparison of responses to trauma-related and nontraumatic stimuli have been the foundation of research in the area, including contrasting responses to psychological and physical stress (e.g., Rouselle, Blascovich, & Kelsey, 1995; Shalev, 1996). Presentation of personalized stimuli in the auditory mode has been proposed as the most favorable medium to investigate psychophysiological responses, although other modes including visual and olfactory have been used (e.g., Blanchard & Buckley, 1999; Keane et al., 1998; McCaffrey, Lorig, Pendrey, McCuthcheon, & Garret, 1993). Personalized imagery, also termed idiosyncratic

stimuli, was pioneered in the traumatology field by Pitman and colleagues (1987), and derived from the work of Lang (1979) on stimulus and response propositions in fear arousing imagery (e.g., Cook, Melamed, Cuthbert, McNeil, & Lang, 1988; Lang, Kozak, Miller, Levin, & McLean, 1980; Lang, Levin, Miller, & Kozak, 1983). Blanchard, Hickling and colleagues have published the most comprehensive series of studies to date of the psychophysiological assessment of PTSD after MVA exposure, following earlier work focusing on Vietnam veterans (e.g., Blanchard, Kolb, Gerardi, & Pallmeyer, 1982), and have utilized personalized imagery to assess posttraumatic responses (e.g., Blanchard & Hickling, 1997; Blanchard, Hickling, Buckley, & Taylor, 1996; Blanchard, Hickling, et al., 1991; Blanchard, Hickling, Taylor, & Loos, 1994b; Blanchard, Hickling, Taylor, Loos, & Gerardi, 1994; Blanchard, Hickling, Buckley, Taylor, Vollmer et al., 1996; Blanchard et al., 1999; Hickling & Blanchard, 1999). They have measured psychophysiological reactivity to MVA related scenes, and endorsed the role of psychophysiological measurement in the assessment and treatment of MVA-related PTSD.

Keane and colleagues (1998) have produced arguably the most comprehensive study of psychophysiological assessment in PTSD to date, reporting the participation of 1461 Vietnam veterans in structured diagnostic interviews, psychometric assessments and psychophysiological assessments. Individuals with current and lifetime PTSD were distinguished from each other, and also individuals with non-PTSD responses to combat trauma when compared on multiple variables. This study has been evaluated as the largest scale to date, and the assessment framework used supports that devised for the series of studies in the present investigation.

Psychophysiological assessment has not been considered a substitute for diagnostic interview, as it provides specific information that requires interpretation in a larger diagnostic framework (e.g., Orr & Kaloupek, 1997). Given that the accuracy of classifying individuals purely on the basis of psychophysiological response is rarely reported to be 100%, this form of assessment for diagnostic purposes may be considered a confirmatory adjunct, rather than a primary diagnostic tool (e.g., Blanchard & Buckley, 1999).

4.2 Summary

No single method of diagnosing PTSD has been considered adequate to assess the complexity of posttraumatic responses. On the basis of the proposed integrated model of the aetiology of posttraumatic stress disorders presented in the previous chapter, it has been considered that multimodal assessment of posttraumatic responses is necessary to comprehensively examine the wide range of variables reported to influence posttraumatic response. Multimodal assessment of posttraumatic responses has been endorsed extensively in the literature (e.g., Carlson, 1996; Keane et al., 1987; Lyons et al., 1988; O'Brien, 1998; Solomon, Keane, et al., 1996; Wolfe et al., 1987), particularly in relation to overcoming the potential effects of response distortion such as malingering. Four empirical studies will now be presented to investigate differential responses to trauma using the example of MVA trauma. These studies have utilized the multimodal assessment types introduced in this chapter, excluding neuroimaging techniques which were not an available resource, but were considered in the directions for further research.

CHAPTER FIVE

STUDY ONE:

THE PSYCHOLOGICAL IMPACT OF MVA EXPOSURE IN AN AUSTRALIAN STUDENT SAMPLE

5.1 Introduction

Despite the fact that MVAs have been reported internationally as being a civilian trauma type with a highly adverse combination of frequency and psychological impact (e.g., Abdel-Aty & Radwan, 2000; Dougall et al., 2001; Friedland & Dawson, 2001; Hobbs & Mayou, 2000; Lally & Sims, 1999; Norris, 1992; Norris, Matthews, & Riad, 2000; Turnbull, 1999), there has been some debate as to whether MVAs should be considered traumatic events (e.g., van der Kolk, 1997). It is likely that this debate has arisen from changes in the DSM criteria for a traumatic event, as earlier definitions excluded events within the realms of common experience. MVAs, due to their frequency, may be considered common experiences. As previously noted, not all MVAs may be defined as psychologically traumatic for the individuals involved, and cultural differences may affect the perception of these types of events as traumatic (e.g., Hayakawa et al., 2000; Kinzie et al., 1998).

Before considering specific posttraumatic diagnoses, it was first considered prudent to establish if MVAs were perceived as traumatic events in the Australian population to be targeted, and that the prevalence of traumatic MVAs was great enough to support an examination of their effects. This study investigates the nature and prevalence of MVA exposure, and associated psychological responses to experiencing MVAs in the target population.

5.1.1 Assessment of the nature of MVAs: Objective versus subjective

Objective assessments of the nature of MVAs have considered factors such as the number of vehicles involved, the nature of physical injuries sustained, the number of people injured or killed in the MVA, the severity of property damage, and third party assessment of threat to life (Australian Bureau of Statistics [ABS], 2001). It has been proposed that these variables may not be adequate indicators of the seriousness of MVAs when considered in isolation from subjective perceptions (e.g., Blanchard & Hickling, 1997). For example, number of deaths may be an objective statistic, but it may not reflect the number of MVAs in which people perceived that they were going to die, or feared the death of others. The DSM-IV (APA, 1994) criteria for posttraumatic stress disorders specify that subjective perceptions of events should be considered more important than objective indicators when assessing the traumatic nature of an event. Subsequently, the assessment of perception of threat to the physical integrity of self or others is required in order to define the traumatic nature of an event (APA, 1994). Both objectively evaluated and subjectively perceived threats to the safety of self and others during trauma have been associated with severity of posttraumatic symptoms (e.g., Blanchard et al., 1997; Pynoos et al., 1987; Thompson et al., 1998).

In terms of the prevalence of MVAs during which fear for the safety of self or others was experienced, inconsistencies have been identified in the collection of this information (see Blanchard & Hickling, 1997). However, it has been reported that the prevalence of fear of death and serious injury experienced in response to traumatic events was higher than objective measures of actual death and injury in MVAs (e.g., McDermott & Cvitanovich, 2000; Stallard, Velleman, & Baldwin,

1998; Thompson, 1999; Udwin, Boyle, Yule, Bolton, & O'Ryan, 2000; Zhao et al., 2001).

5.1.2 Posttraumatic symptoms in response to MVAs

As has been previously established, posttraumatic symptoms have been widely reported to result from exposure to MVAs (e.g., Dougall et al., 2001; Friedland & Dawson, 2001; Fullerton et al., 2000; Ho, Davidson, Van Dyke, & Agar-Wilson, 2000; Hobbs & Mayou, 2000; McDermott & Cvitanovich, 2000). One example of the prevalence of posttraumatic symptoms following MVAs has been reported by Blanchard and Hickling (1997) using data from the Albany MVA Project. The data were obtained using a cohort of survivors of serious MVAs (N = 158) with a mean age of 35.4 years, and 32% of the sample were male. The sample was divided into the following groups on the basis of reported symptoms: PTSD (n = 62), subsyndromal PTSD (n = 45), and non-PTSD (n = 51). The most commonly reported symptoms in the three groups were distressing reminders of the event (PTSD and subsyndromal groups), and sleep disturbance (non-PTSD group). It was considered likely that non-patient populations exposed to serious MVAs, even those individuals without a posttraumatic stress disorder diagnosis, would report experiencing posttraumatic symptoms to some degree.

5.1.3 Sex and age differences in MVA exposure and posttraumatic symptoms

Road safety statistics have indicated that male drivers have been more frequently involved in fatal and serious MVAs than female drivers (e.g., Attewell, 1998; ABS, 1996; Dobson, Brown, Ball, Powers, & McFadden, 1999; Evans,

1991; Massie, Green, & Campbell, 1997). This sex difference has been attributed to various factors, including the propositions that females drive less than males, and males are more likely to engage in risk taking behaviours such as speeding and driving under the influence of alcohol (e.g., Abdel-Aty & Radwan, 2000; Laapotti & Keskinen, 1999; Weber, 1975; Wylie, 1995). The United States National Centre for PTSD (Buckley, 2000) reported that recent statistics have shown that accidents have been experienced by 25% of males and 13% of females in the USA during their lifetime. These statistics were also presented by Blanchard and Hickling (1997), however, they noted that the study from which the data were extracted (Kessler et al., 1995) may have only included accidents considered to be traumatic. Blanchard and Hickling stated that the majority of Americans are likely to have experienced at least one minor MVA by 30 years of age.

Regardless of sex, young drivers (18-24 years old) have been documented as a high risk cohort for involvement in MVAs (e.g., Dobson et al.,1999; Hijar, Carrillo, Flores, Anaya, & Lopez, 2000; Laapotti & Keskinen, 1999; McGwin & Brown, 1999; Norris et al., 2000; Peltzer, 1999; Waller, Elliott, Shope, Raghunathan, & Little, 2001). The risk has been associated with driver inexperience, risk-taking behaviour, and lifestyle factors (e.g., Abdel-Aty & Radwan, 2000; Evans & Wasielewski, 1983; Finn & Bragg, 1986; Gregersen & Berg, 1994; Jessor, 1987; Jonah, 1986; Matthews & Moran, 1986; Michiels & Schneider, 1984; Laapotti & Keskinen, 1999; Summala, 1987). Older drivers, defined as over 65 years of age, have also been documented as a high risk cohort for involvement in MVAs, and factors such as diminished sensory abilities, functional impairment, chronic medical conditions and reduced response times have been proposed to be contributing factors (e.g., Abdel-Aty & Radwan, 2000;

McGwin & Brown, 1999). Youngest and oldest drivers are reported to be more likely to be found at fault in MVAs than middle-aged drivers (McGwin & Brown, 1999). When considering both sex and age, young males have been found to be the highest risk cohort for involvement in an MVA (e.g., Lang & Stockwell, 1991; Simon & Corbett, 1996; Steensburg, 1994).

Sex and age differences in vulnerability to the psychological impact of trauma have been reported, including reports of higher prevalence of the diagnosis of PTSD in females (e.g., Allden et al., 1996; Breslau, Davis, Andreski, Peterson, & Schultz, 1997; Green et al., 1997; Lowenstein, 2001; Pennebaker, 2000; Pereira, 1999; Seedat & Stein, 2000). Females have been reported to be more likely to develop PTSD, but less likely to be exposed to traumatic events than males (e.g., Breslau et al., 1991; Norris, 1992; Vrana & Lauterbach, 1994). It has been noted that response biases may affect investigations of sex differences, due to the considerable reliance on self-report describing posttraumatic symptoms, and that males may be more likely to under-report the presence and severity of symptoms (e.g., Deahl, Srinivasan, Jones, Neblett, & Jolly, 2001). The DSM-IV (APA, 1994) diagnostic classifications of PTSD and ASD specify differences in posttraumatic symptomatology between children and adults. However, the diagnostic criteria do not specify sex or age differences in adult posttraumatic responses.

5.1.4 The prevalence and nature of MVAs in Tasmania

MVAs have been reported to be a major cause of death and injury in Australia, and have been said to incur costs in excess of $AUS15 billion annually (ABS, 2001). Since MVA death records commenced in Australia in 1925, it has been reported that 164,190 people have been killed in MVAs, which is almost

double the number of Australians killed in the four major wars in which Australia was involved in the 20th century (89,850 deaths) (ABS, 2001). Table 3 displays the number of people killed in MVAs in each state and territory of Australia in the last ten years (1991-2000) according to the ABS (2001).

Table 3.

Number of people killed in MVAs in each state and territory of Australia in the last ten years (1991-2000) (ABS, 2001).

Year	NSW	VIC	QLD	SA	WA	TAS	NT	ACT	Total
1991	663	503	395	184	207	77	67	17	2113
1992	649	396	416	165	200	74	54	20	1974
1993	581	435	396	218	209	58	44	12	1953
1994	646	378	418	159	211	59	41	17	1928
1995	620	418	456	181	209	57	61	15	2017
1996	581	417	385	181	247	64	72	23	1970
1997	576	377	361	148	197	32	60	17	1768
1998	556	390	279	168	223	48	69	22	1755
1999	578	381	313	151	217	53	49	19	1761
2000	603	405	317	166	213	43	52	18	1817

Key: NSW = New South Wales, VIC = Victoria, QLD = Queensland, SA = South Australia, WA = Western Australia, TAS = Tasmania, NT = Northern Territory, ACT = Australian Capital Territory.

As noted in table 3, there are six states and two territories of Australia, and the present investigation focused on MVAs experienced by a sample of the

population in Tasmania. These statistics indicated that fewer people have been killed in MVAs in Tasmania than all other states and territories except the Australian Capital Territory. However, these statistics may be considered misleading because, when fatalities per 100,000 persons in the population were calculated, Tasmania had the second highest rate of deaths (ABS, 2001). Tasmania has the smallest population of the Australian states, recorded as 470,400 people according to the most recent statistics, representing approximately 2.5% of the total Australian population (ABS, 2001).

The main causes of injury in MVAs in Tasmania have been reported to be driver inattention, speed, failure to give way, and road conditions (Department of Infrastructure, Energy and Resources [DIER], 2001). From the years 1996 to 2000, approximately 4% of the population of Tasmania (N = 18,816) were involved in MVAs resulting in death or injury. The DIER (2001) reported that of those involved, 61% of the individuals killed were drivers, 25% were passengers, and 14% were pedestrians. In the same time frame, it has been reported that 63% of the individuals injured were drivers, 29% were passengers, and 8% were pedestrians. The majority of deaths occurred in non-residential areas (71%), and the majority of injuries occurred in residential areas (62%). In terms of age and sex high risk cohorts, males aged between 17 and 29 were reportedly at greatest risk of dying or being injured in an MVA. Figure 2 is a map of Tasmania showing the occurrence of MVA deaths in 1999, the most recent annual statistics of this type available at the time of writing, as recorded by the Royal Automobile Club of Tasmania [RACT] (2000).

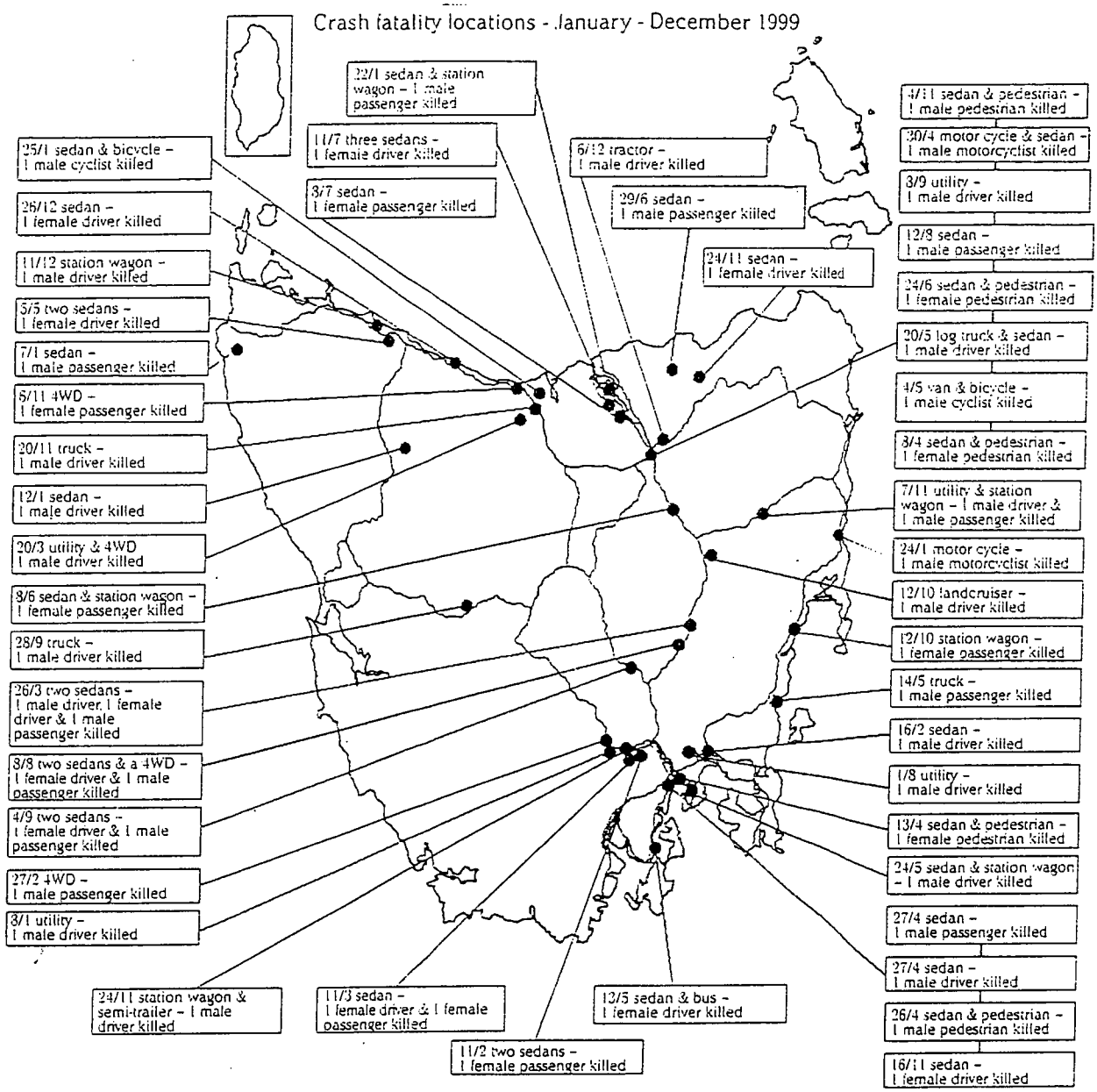

Figure 2.

Map of Tasmania showing the locations of MVA deaths in 1999 (RACT, 2000).

5.1.5 Aims and hypotheses

This study investigated the prevalence of MVAs and associated posttraumatic stress symptoms in a sample of university students in Tasmania. It was deemed necessary to establish that there was considerable prevalence of MVA exposure reported by this population, and that a sufficient number of MVAs were perceived as traumatic, to warrant further study. This study provided introductory data regarding the prevalence and nature of MVAs in this Australian sample, and examined sex differences in the nature and prevalence of MVA exposure and reported psychological symptoms. On the basis of the reviewed literature, it was hypothesized that:

1. The majority of the participants were expected to have experienced at least one minor MVA.

2. Reports of fear of death and injury in response to MVAs were expected to exceed reports of actual death and injury.

3. Each posttraumatic symptom type was expected to be reported by the subgroup of participants exposed to MVAs.

4. Approximately 25% of males and 13% of females in the sample were expected to have experienced a traumatic MVA.

5. A greater proportion of females than males were expected to report posttraumatic symptoms.

5.2 Method

5.2.1 Participants

Participants were 425 students from the University of Tasmania. Participation was voluntary, and anonymous if desired. The mean age of participants was 20.10 years (SD = 7.20), with a median age of 19 years, and ranged from 17 to 50 years. The sample (N = 425) consisted of 330 females (78% of sample) and 95 males.

5.2.2 Materials

The Motor Vehicle Accident Questionnaire [MVAQ] was designed for this study (Holmes, 1997a), and is presented in Appendix B-1. The MVAQ was designed to investigate the prevalence and nature of MVAs involving the student population, and provide a brief screening assessment of psychological responses to the events. The MVAQ consisted of 16 items eliciting information about participant demographics, history and nature of involvement with MVAs, recall of peritraumatic responses, and posttraumatic symptoms. The symptom items represented three of the diagnostic criteria within each of the four major symptom clusters of PTSD and ASD (reexperiencing, avoidance and numbing, dissociation and hyperarousal), in order to provide a representative overview of symptom type prevalence in a brief screening format. The MVAQ was designed to assess the presence/absence of posttraumatic symptoms and peritraumatic fear in a simple dichotomous format, as these variables are examined in more detail in the next three studies. Should respondents have been involved in more than one MVA, they were instructed to answer the questions in relation to the MVA that they felt was

the most distressing psychological experience for them. In addition to providing information about MVA exposure in the sample, the MVAQ was also designed to identify potential participants for subsequent studies as part of the proposed non-patient community sample.

5.2.3 Procedure

Ethics approval was obtained prior to data collection from the University of Tasmania Research Ethics Committee. Five hundred students attending a psychology lecture at the University of Tasmania were invited to participate. The MVAQ was distributed at the end of the lecture. Students were asked to complete the questionnaire and return it to the researcher before departure. No time limit was applied, and the researcher was available to answer enquiries.

5.2.4 Design and data analysis

The study had a survey based design. Responses to the items of the MVAQ were the dependent variables of the study. Responses of the total sample were collated and descriptive statistics calculated. T-tests, Cochran Q tests, and chi square analyses were used for data comparisons.

5.3 Results

5.3.1 Overview

The prevalence and nature of MVA exposure in the sample were presented, including consideration of both objective and subjective factors. Prevalence rates of peritraumatic fear of death and serious injury were then presented. Posttraumatic

symptoms reported by the sample in response to MVAs were presented for each DSM-IV posttraumatic symptom cluster (reexperiencing, avoidance and numbing, hyperarousal and dissociation). Sex differences in all of the above measures were then examined.

5.3.2 MVA details

The frequency data with conversion into percentages for MVAQ items are presented in Table 4, with reference to the subgroup of the sample who reported exposure to an MVA (n = 247). The response rate was 85%, with 425 of the 500 questionnaires returned completed. Over half of the participants (58%) reported that they had been involved in an MVA, with not all MVAs necessarily occurring in Tasmania. One third of the total sample (33%) reported that they had been involved in a traumatic MVA that caused them to feel intense emotions such as fear, helplessness or horror (APA, 1994). Of the participants who had been exposed to an MVA, over half reported the experience to have been psychologically traumatic. Mean time elapsed since the MVA was 2.32 years (SD = 8.45), and ranged from one week to 12 years. The majority of participants were passengers in MVAs, and a very small minority were pedestrians. The majority of MVAs involved two vehicles, and property damage only.

Approximately one third of the MVAs involved injury to one or more people. One participant had been involved in an MVA in which three people were killed, but no other fatal accidents were reported. Of the 247 participants who had been involved in an MVA, one quarter reported that they had been injured in the accident. Of the injuries reported, 40% were described as cuts and bruising; 35% broken bones and sprains; 15% whiplash and concussion; and 10% were described

as internal injuries. Participants who reported involvement in an MVA were significantly older at the time of assessment (M = 23.0, SD = 8.20 years) than those who reported no involvement in an MVA (M = 20.2, SD = 4.92 years), t (1, 246) = 4.00, p <.0001.

Table 4.

Frequency data with conversion into percentages for MVA detail items of the MVAQ (n = 247).

MVAQ item		*Freq.*	(%)
MVA details			
Exposure to a traumatic MVA		141	(57)
Role in MVA	Driver	114	(44)
	Passenger	131	(55)
	Pedestrian	2	(1)
No. of vehicles in MVA	One	79	(32)
	Two	154	(62)
	Three +	14	(6)
No. people injured in MVA	Nil	167	(68)
	One	42	(16)
	Two	19	(8)
	Three +	19	(8)
Self injured in MVA (Yes)		61	(25)

5.3.3 Fear of death and serious injury

As previously noted, not all participants who had been exposed to MVAs perceived them to be psychologically traumatic. However, for the proportion who did experience psychological trauma, fear of death and/or serious injury in response to the event was reported. Table 5 displays the frequency data with conversion into percentages for the sample exposed to MVAs who reported fear of death and serious injury.

Cochran Q tests were employed to assess the significance of differences in within-group frequencies on the multiple dichotomous variables. It was found that the sample exposed to MVAs more frequently reported: fear of serious injury than the death of others involved in the MVA, Q (1,247) = 62.06, $p < .0001$; and fear of serious injury to themselves rather than their own death resulting from the MVA, Q (1,247) = 53.00, $p < .0001$.

Table 5.

Frequency data with conversion into percentages for the sample exposed to MVAs reporting fear of death or serious injury (n = 247).

MVAQ item (Yes)	*Freq.*	(%)
At the time of the accident, did you fear that you were going to die?	30	(12)
Did you fear that someone else involved was going to die?	44	(18)
Did you fear that you were going to be seriously injured?	84	(34)
Did you fear that someone else was going to be seriously injured?	114	(46)

It was also found that fear of someone else dying in the MVA was more frequently reported than fear of own death occurring in the MVA, Q (1,247) =

6.74, $p = .009$. Fear for the safety of others over self was also evident in terms of serious injury with fear for others more frequently reported than fear of serious injury occurring to self in the MVA, $Q\ (1,247) = 12.79, p = .0003$.

5.3.4 Posttraumatic symptoms

Table 6 displays the percentage of participants who had experienced an MVA and reported posttraumatic symptoms following the event. Results were analyzed using Cochran Q tests to examine the most frequently reported symptom within each cluster.

Table 6.

Frequency data with conversion into percentages for the sample reporting posttraumatic symptoms following MVA exposure (n = 247).

MVAQ symptom item	*Freq.*	(%)
Reexperiencing symptoms		
1. Distressing dreams	22	(9)
2. Distressing flashbacks	36	(15)
3. Distressing reminders of the accident	64	(26)
Avoidance and numbing symptoms		
4. Trying to avoid reminders	46	(19)
5. Trying to avoid thinking/talking about the MVA	33	(13)
6. Feeling flat and unable to react emotionally	30	(12)

(Table continued...)

Table 6 (continued...)

MVAQ symptom item	*Freq.*	(%)
Hyperarousal symptoms		
7. Sleeping and/or concentration difficulties	24	(10)
8. Increased irritability or anger	31	(13)
9. Feeling jumpy	71	(29)
Dissociative symptoms		
10. Feeling in a daze	57	(23)
11. Feeling numb	39	(16)
12. Feeling unreal or detached	57	(23)

For ease of reference, each symptom will be referred to in this section using an abbreviation for data comparisons (e.g., symptom 1 = S1). Cochran Q tests demonstrated that within each symptom cluster, some symptoms were reported more frequently than others. In terms of reexperiencing symptoms, distressing reminders of the MVA (S3) were found to be more frequently reported than distressing flashbacks (S2), and both symptoms were more frequently reported than distressing dreams (S1), S1 vs S2, Q (1, 247) = 4.50, p = .03; S2 vs S3, Q (1, 247) = 13.25, $p < .0001$; S1 vs S3, Q (1, 247) = 26.68, $p < .0001$.

In relation to avoidance and numbing symptoms, trying to avoid reminders of the MVA (S4) was reported significantly more frequently than both trying to avoid thinking/talking about the MVA (S5) and feeling flat and unable to respond emotionally (S6), S4 vs S5, Q (1, 247) = 4.67, p = .03; S5 vs S6, Q (1, 247) = 1.45, $p > .05$; S4 vs S6, Q (1, 247) = 8.07, p = .005.

With respect to hyperarousal symptoms "feeling jumpy" (S9) was reported significantly more frequently than both sleeping/concentration difficulties (S7) and increased irritability or anger (S8), S7 vs S8, Q (1, 247) = 0.64, $p > .05$; S8 vs S9, Q (1, 247) = 18.28, $p < .0001$; S7 vs S9, Q (1, 247) = 22.22, $p < .0001$. Finally, in terms of dissociative symptoms, both feeling in a daze (S10) and feeling unreal or detached (S12) were more frequently reported than feeling numb (S11), S10 vs S11, Q (1, 247) = 11.31, $p = .0007$; S11 vs S12, Q (1, 247) = 6.56, $p = .01$; S10 vs S12, Q (1, 247) = .07, $p > .05$.

In order to further investigate the experience of specific posttraumatic symptom types, chi square analyses were used to compare responses between the subgroup reporting that the MVA was traumatic (n = 141), versus those who reported that the experience was not traumatic (n = 106). The responses of the nontraumatic MVA subgroup are summarized in Appendix B-2, given that the responses of individuals exposed to traumatic MVAs are the focus of the present investigation.

Participants reporting that the MVA was traumatic described a significantly greater prevalence of nine of the twelve symptoms [distressing dreams: χ^2 (1, N = 247) = 14.79, $p < .0001$; distressing flashbacks, χ^2 (1, N = 247) = 22.50, $p < .0001$; distressing reminders of the MVA, χ^2 (1, N = 247) = 19.43, $p < .0001$; trying to avoid reminders, χ^2 (1, N = 247) = 13.36, $p < .0001$; trying to avoid thinking/talking about the MVA, χ^2 (1, N = 247) = 10.29, $p < .0001$; sleeping and/or concentration difficulties, χ^2 (1, N = 247) = 5.39, $p < .05$; increased irritability or anger, χ^2 (1, N = 247) = 6.85, $p < .01$; feeling jumpy, χ^2 (1, N = 247) = 5.12, $p < .05$; feeling unreal or detached, χ^2 (1, N = 247) = 19.06, $p < .0001$].

There was no significant difference in the reported experience of the following three symptoms between the subgroups: feeling flat and unable to respond emotionally, χ^2 (1, N = 247) = 1.45, p >.05; feeling in a daze, χ^2 (1, N = 247) = 4.83, p >.05; and feeling numb, χ^2 (1, N = 247) = 4.73, p >.05.

Describing the MVA as traumatic was found to be significantly more frequently reported by participants fearful of the death of others in the MVA, χ^2 (1, N = 247) = 18.18, p < .0001; participants fearful of their own death in the MVA, χ^2 (1, N = 247) = 15.44, p < .0001; participants fearful of serious injury to others during the MVA, χ^2 (1, N = 247) = 20.80, p < .0001; and participants fearful of sustaining serious injuries to themselves during the MVA, χ^2 (1, N = 247) = 26.55, p < .0001. Describing the MVA as traumatic was found not to be significantly related to current age, t (246) = 0.44, p >.05; age at the time of the MVA, t (246) = 1.23, p < .05; number of vehicles involved in the MVA, t (246) = 1.04, p >.05; number of people injured in the MVA, t (246) = 0.86, p >.05; being injured in the MVA, t (246) = 3.43, p >.05; and role in the MVA, t (246) = 0.60, p >.05.

5.3.5 Sex differences

Responses to the MVAQ were analyzed in order to investigate sex differences. Significantly more males (69%) than females (55%) in the total sample reported that they had been involved in an MVA, χ^2 (1, N = 425) = 5.60, p < .01. However, significantly more females (36%) than males (24%) reported exposure to a traumatic MVA, χ^2 (1, N = 425) = 6.75, p < .01. There were no significant differences between males and females in terms of current age, t (424) = 1.69, p > .05; or age at which the MVA occurred, t (424) = .98, p > .05. Table 7 displays

frequency data with conversion into percentages and chi square analyses of the MVAQ responses for male and female participants reporting MVA exposure.

Table 7.

Frequency data with conversion into percentages and chi square analyses of MVAQ item responses for male (n = 66) and female (n = 181) participants reporting involvement in an MVA.

MVAQ item		Males	Females		
		Freq. (%)	*Freq.* (%)	χ^2 *(df)*	*p*
MVA details					
Traumatic MVA	Yes	23 (35)	118 (65)	10.92 (1)	< .001
Role in MVA	Driver	43 (65)	71 (39)	11.17 (2)	< .01
	Passenger	22 (33)	109 (60)		
	Pedestrian	1 (1)	2 (1)		
No. of vehicles in MVA	One	26 (39)	52 (29)	.75 (2)	n.s.
	Two	35 (53)	119 (66)		
	Three +	5 (8)	9 (5)		
No. people injured in MVA	Nil	43 (65)	125 (69)	.90 (3)	n.s.
	One	16 (24)	25 (14)		
	Two	4 (6)	14 (8)		
	Three +	3 (5)	16 (9)		
Self injured in MVA	Yes	15 (23)	45 (25)	.19 (1)	n.s.

(Table continued...)

Table 7 (continued...)

MVAQ item	Males	Females		
	Freq. (%)	*Freq.* (%)	χ^2 (*df* = 1)	*p*
Fear of death and injury				
Fear of own death	6 (9)	24 (13)	.62	n.s.
Fear of other death	8 (12)	36 (20)	2.14	n.s.
Fear of own injury	25 (38)	60 (33)	.02	n.s.
Fear of other injury	28 (42)	87 (48)	.53	n.s.
Posttraumatic symptoms				
Distressing dreams	3 (5)	18 (10)	1.90	n.s.
Distressing flashbacks	8 (12)	27 (15)	.58	n.s.
Distressing reminders of the accident	12 (18)	52 (29)	5.25	< .05
Trying to avoid reminders	5 (8)	42 (23)	7.05	< .01
Trying to avoid thinking/talking about it	8 (12)	25 (14)	.43	n.s.
Feeling flat/unable to react emotionally	8 (12)	22 (12)	1.47	n.s.
Sleeping and/or concentration difficulties	3 (5)	22 (12)	4.28	< .05
Increased irritability or anger	10 (15)	22 (12)	.45	n.s.
Feeling jumpy	14 (21)	56 (31)	.73	n.s.
Feeling in a daze	16 (24)	42 (23)	.01	n.s.
Feeling numb	12 (18)	27 (15)	.25	n.s.
Feeling unreal or detached	11 (17)	45 (25)	.93	n.s.

The description of the MVA as traumatic was found to be reported significantly more frequently by females than males. There was a significant sex difference in role played in the MVA, with the majority of females being a

passenger, and the majority of males driving during the MVA. No sex differences were found in terms of the number of vehicles involved in the MVA, the number of people injured, or self being injured. There were also no sex differences found in the reported prevalence and nature of fear of death and injury. In terms of posttraumatic symptoms, sex differences were found for three of the twelve symptoms. Distressing reminders of the MVA, trying to avoid reminders, and sleeping and/or concentration difficulties were reported by significantly more females than males.

5.4 Discussion

5.4.1 Nature and prevalence of MVA exposure

In support of the first hypothesis, the majority of the sample reported exposure to at least one MVA. These findings are consistent with the statistics previously presented that suggest Tasmania has a high rate of MVAs per population when compared with other states and territories of Australia (ABS, 2001). This finding also concurs with Blanchard and Hickling's (1997) statement relating to the American population, that the majority would have experienced an MVA by age 30. Of those participants reporting involvement in an MVA, the majority reported the experience to be psychologically traumatic.

The fact that a majority of the sample described the MVA as psychologically traumatic conflicts with the proposal that only a minority of MVAs, at the most severe end of the objective spectrum, should be considered traumatic events (e.g., van der Kolk, 1997). It may be speculated that, despite the

clarity of the DSM-IV (APA, 1994) definition of a traumatic event, subjective assessments of what constitutes a traumatic stressor may be considered variable.

The MVAs experienced by participants were most often two vehicle MVAs involving property damage only, and most participants were passengers in the MVAs. The data reflected that individuals who reported the MVA to be psychologically traumatic were not necessarily exposed to accidents involving death or serious injury. This finding may be considered to have implications for the impact of perceived threat, versus actual death or injury, in the experience of psychological trauma. As reviewed in the literature, both actual and perceived threat have been associated with severity of posttraumatic symptoms (e.g., Blanchard et al., 1997; Pynoos et al., 1987; Thompson et al., 1998). As would be expected, history of exposure to MVAs was found to be associated with increasing age.

5.4.2 Fear of death and serious injury

In support of the second hypothesis, fear of death and serious injury exceeded reports of actual death and injury. The participants most commonly reported fear for the safety of others, rather than themselves. This fear has been recognised in the DSM-IV definition (APA, 1994) of a traumatic event, which indicates that perceived threat to others can be psychologically traumatic. Fear of serious injury was more frequently reported than fear of death. It may be speculated that this finding is reflective of the relatively low level of seriousness of the MVAs as objectively assessed and the actual low likelihood of death occurring, or it may be a manifestation of concern for physical integrity despite the belief that

death is not imminent (e.g., McDermott & Cvitanovich, 2000; Stallard et al., 1998; Thompson, 1999; Udwin et al., 2000; Zhao et al., 2001).

5.4.3 Posttraumatic symptoms

With respect to posttraumatic symptoms, all reexperiencing, avoidance and numbing, hyperarousal and dissociative symptoms were reported in response to MVA exposure, in support of the third hypothesis. Distressing reminders of the MVA, feeling 'jumpy', feeling in a daze, and feeling unreal or detached from surroundings were the most commonly reported symptoms. Trying to avoid reminders of the MVA was the most frequently reported symptoms from the avoidance and numbing cluster. Only three MVA exposed participants reported being symptom-free, and they had not considered the MVA to be psychologically traumatic. These findings support previous research using American and English samples, in terms of the prevalence of symptom types and the observation that the majority of individuals exposed to a traumatic MVA report experiencing at least one type of posttraumatic symptom following the event (e.g., Blanchard & Hickling, 1997; O'Brien, 1998). The fact that only three participants exposed to MVAs reported being symptom-free, emphasized the psychological impact of MVAs, whether or not they were perceived to be traumatic.

When only the MVAs perceived as traumatic were considered, every individual in this subgroup reported experiencing at least one posttraumatic symptom in response to the MVA. The results indicate that this subgroup reported a significantly greater prevalence of all symptoms, excluding the avoidance symptom feeling flat and unable to respond emotionally, and the dissociative

symptoms, feeling numb and in a daze. It may be speculated that these symptoms are reflective of adaptive self-protection processes.

Perceiving the MVA as traumatic was found not to be associated with objective variables such as current age or age at the time of the MVA, number of vehicles involved, number of people injured, being injured, or role in the MVA. These findings support the DSM-IV (APA, 1994) emphasis that subjective rather than objective indicators are most relevant in determining the traumatic nature of an MVA.

5.4.4 Sex differences

The fourth hypothesis was only partially supported. Although the findings were consistent with the literature in that significantly more males than females reported exposure to an MVA (e.g., Attewell, 1998; ABS, 1996; Dobson et al., 1999; Evans, 1991; Massie et al., 1997), and significantly more females than males reported exposure to a traumatic MVA (e.g., Keane, 1998), the findings indicated that 36% of the females in the sample reported exposure to traumatic MVAs, which exceeded the expected prevalence of 13% suggested by the United States National Centre for PTSD statistics (Buckley, 2000). The male prevalence rates were comparable, with 25% reported in the American statistics, and 24% found in this study. Therefore, Australian females in this sample were found to be of almost three times greater risk of exposure to a traumatic MVA than their American counterparts. This finding also conflicts with previous statements that females are less likely to be exposed to traumatic events than males (e.g., Breslau et al., 1991; Norris, 1992; Vrana & Lauterbach, 1994).

It may be speculated that the females in this sample were exposed to higher risk travel or more frequent travel by road than other females. As most females were passengers, it may have been role in the MVA, rather than sex, that was the more influential factor in psychological response. For example, the sense of loss of control experienced by a passenger, and the attribution of blame to another person, have been associated with the severity of posttraumatic symptoms (e.g., Hickling, Blanchard, Buckley, & Taylor, 1999; Ho et al., 2000; Wells, Haines & Williams, 2000).

No sex differences were found in objective MVA variables, such as the number of vehicles involved or injuries caused, and no sex differences were found in the reported prevalence and nature of peritraumatic fear. These results attested to the comparability of the severity of the MVAs, despite the fact that male drivers have been reported to have different types of MVAs to female drivers (Panek & Rearden, 1987), and despite the differences between males and females in this sample in terms of their role in the MVA.

In partial support of the fifth hypothesis, females were found to endorse three of the twelve posttraumatic symptoms significantly more frequently than males. Distressing reminders of the MVA, trying to avoid reminders of the MVA, and sleeping/concentration difficulties were reported by significantly more females than males. It is unclear as to whether females were more likely to report certain symptoms than males, or if the data truly reflects the greater experience of symptoms by females. Literature regarding Australian culture has suggested that some males, particularly in this age cohort, may be less likely to report emotional or psychological symptoms due to stoic views of masculinity (e.g., Lynski, Degenhardt, & Hall, 2000; Theodore & Lloyd, 2000). Interpretations are

complicated by the skewed sample, being predominantly female, and cannot be further investigated from the available data. However, it may be useful to note that although there is little evidence in the literature of sex differences in intrusive and avoidance symptoms, in general, females have been found to have higher arousability than males and this has translated into an increased likelihood of sleep disturbance for females (Coren, 1990). Reports have indicated that a considerably greater number of females than males (41.7% versus 29.9% respectively) experienced insomnia (Husby & Lingjaerde, 1990) and that this was particularly the case with females with some form of psychopathology such as depression (Zetin, Sklansky, & Cramer, 1984). Other research has reported that in response to traumatic loss of a significant other, college ages females were more likely to experience insomnia than male cohorts (LaGrand, 1985).

5.4.5 Summary and conclusions

The majority of this sample of Australian university students reported exposure to at least one MVA. The majority of the sample who had been exposed to an MVA reported the experience to be psychologically traumatic. Exposure rates were consistent with previous American data (e.g., Blanchard & Hickling, 1997), and conflicted with the suggestion that only a minority of MVAs at the most severe end of the objective spectrum could be considered traumatic events (e.g., van der Kolk, 1997).

Fear of death and serious injury was found to exceed objective measures of MVA-related injuries and deaths, having implications for the severity of posttraumatic stress symptoms. This finding has been supported by the DSM-IV criteria (APA, 1994), and other studies (e.g., McDermott & Cvitanovich, 2000;

Stallard et al., 1998; Thompson, 1999; Udwin et al., 2000). The full spectrum of posttraumatic symptoms was reported within the sample, supporting previous research using American and English samples. Subjective, rather than objective MVA variables, were found to be associated with posttraumatic symptom outcomes.

Males in the sample were found to have comparable exposure rates to MVAs with other studies, however, females in the sample were found to have almost three times greater risk of exposure to a traumatic MVA than American females. This finding was interpreted in terms of potentially mediating variables, such as the result relating more to being a passenger in the MVA than being female. Sex differences in the report of posttraumatic symptoms were also discussed. It was determined from the findings that traumatic MVA exposure occurred with sufficient frequency in this population to make a series of studies of diagnostically distinct posttraumatic responses to MVA trauma a viable option. Additional recruitment from the wider community was also deemed desirable for subsequent studies in order to examine the psychological impact of MVAs in a more heterogeneous sample.

Given that it was indicated in the findings of this study that females reported greater prevalence of posttraumatic symptoms and exposure to traumatic MVAs, it was predicted that there would be more females than males in the diagnostic groups used for the following three studies. Blanchard and Hickling (1997) reported that research focusing on the aetiology of posttraumatic stress disorders following MVAs has examined three sets of variables: characteristics of the individual that were present prior to the MVA, peritraumatic variables and post-accident variables. The next three studies utilize this approach, and provide an

examination of biopsychosocial variables and their relationships to the development of distinct posttraumatic psychological responses.

This study has been previously presented in part, and referenced contributions by the author of this thesis are underlined:

Haines, J., Williams, C.L., Holmes, G.E., & Mycak, M. (2000, April) *Psychological and psychophysiological responses to motor vehicle accident trauma and the peritraumatic death imprint.* Paper presented at the 3rd World Congress of the International Society of Traumatic Stress Studies, Melbourne, Australia.

Haines, J., Williams, C.L., Mycak, M., & Holmes, G.E. (2000, February). *Psychophysiological responses to motor vehicle accident trauma and the peritraumatic death imprint.* Paper presented at the 10th Conference of the International Organization of Psychophysiology, Sydney, Australia.

Haines, J., Williams, C.L., Holmes, G.E., Wells, J.H., & Mycak, M. (2001). Peritraumatic death imprint: Prevalence, correlates, and determinants of positive and negative outcomes *(manuscript submitted for review).*

CHAPTER SIX

STUDY TWO:

COPING STYLES AND PERSONAL BELIEFS ASSOCIATED WITH THE DEVELOPMENT OF POSTTRAUMATIC STRESS DISORDERS

6.1 Introduction

As discussed in chapter three, individuals enter a traumatic experience with predisposing biopsychosocial characteristics that, regardless of the nature of the trauma and posttrauma environment, may influence the course of posttraumatic psychological recovery (e.g., Paris, 2000). Two variables, coping styles and personal beliefs, were selected for examination in this study to investigate their role in the development of diagnostically distinct responses to MVA trauma. These variables were selected from the many biopsychosocial variables proposed to affect posttraumatic outcomes, due to their reported influence on a range of cognitive, affective, behavioural and biological outcomes (e.g., Felton, 2001; Rosenberg, 2001; Stallard, Velleman, Langsford, & Baldwin, 2001).

Cognitive-behavioural treatments for posttraumatic symptoms have focused on the acquisition and maintenance of adaptive coping skills and on the belief systems that underlie an individual's behaviour, expectations and appraisal of traumatic events (Sherman, 1998). This approach has provided recognition that the development and maintenance of posttraumatic symptoms may be mediated by these variables (e.g., Blanchard, Hickling, Taylor, & Loos, 1995; Falsetti, 1997; Tunnecliffe & Tunnecliffe, 1997). The introduction of the diagnosis of ASD presented a unique opportunity to profile the coping styles and belief systems of individuals with a non-PTSD posttraumatic diagnosis, with the aim of identifying cognitive-behavioural factors that may be targeted in the early assessment and treatment of posttraumatic responses. It was proposed that if cognitive-behavioural factors could be identified which distinguished individuals diagnosed with ASD from those with PTSD and subclinical diagnoses, then ASD as a distinct diagnostic

entity may be better understood. It was also considered that knowledge may be gained about the role of adaptive processes in facilitating more effective recovery from posttraumatic symptoms.

6.1.1 Coping styles and posttraumatic psychopathology

Adaptive coping styles have been attributed with powers of inoculation against the development of posttraumatic symptoms and, conversely, maladaptive coping styles have been associated with the development and maintenance of posttraumatic symptoms (e.g., Fontana & Rosenheck, 1998; Griffing, 1998; Livneh, Antonak, & Gerhardt, 2000; van der Kolk, McFarlane, et al., 1996). The processes underlying coping styles, therefore, have been considered important moderators of life event stress-psychopathology relationships and posttraumatic behavioural outcomes (Hovanitz & Kozora, 1989).

Coping styles have been conceptualised and categorised in various forms that encompass specific coping strategies to define a particular coping style (e.g., Aldwin & Levenson, 1987; Moos & Billings, 1982), with little agreement on a uniformly accepted typology evident in the literature. However, most models have proposed that there are coping styles that are directed at altering three elements: the stressful situation (behavioural strategies), emotional response to the situation (affective strategies), and interpretation of the situation (cognitive strategies). The three types of styles have been reported to be used in isolation and in combination (e.g., Kessler, Price, & Wortman, 1985).

One example of a classification of coping styles that combined these three elements was that of Winje and Ulvik (1995). They proposed the following five themes said to represent aspects of adaptive coping with trauma:

1. Cognitive coping: The need to know what happened;
2. Emotional coping: The need to express emotions;
3. Social coping: The need to share thoughts and feeling with others;
4. Behavioural coping: The need to respond according to the new situation;
5. Existential coping: The need to re-establish meaning regarding life and death.

This model highlighted specific cognitive-affective and behavioural strategies that were proposed to moderate posttraumatic psychological responses. General factors found across several instruments designed to measure coping styles have been reported to be problem engagement, avoidance and social/emotional strategies (e.g., Cook & Heppner, 1997). Problem focused and emotion focused strategies have been proposed to have differential effects on psychological adjustment, with contradiction in the literature regarding the effectiveness of each type of strategy (e.g., Baum, Fleming, & Singer, 1983; Menaghan, 1982; Mitchell & Hodson, 1983).

Psychometric tools such as the Coping Strategies Inventory [CSI] (Tobin, Holroyd, & Reynolds, 1984), have been employed to investigate the structure of coping styles of individuals exposed to stressful events by assessing self-report of the use of specific strategies when dealing with a stressful event (e.g., Livneh et al., 2000; Rainey, 1998; Shriner, 1999; Willert, 1996). Such tools have been adopted to investigate whether differences in psychological adjustment following exposure to stressful events can be accounted for by differences in the use of various types of coping strategies, and overall coping styles. In one example of such research, survivors of sexual assault with more severe psychological symptoms were found to report higher levels of problem focused disengagement coping styles than survivors with less severe symptoms (Shriner, 1999). Maladaptive strategies, such

as self criticism, social withdrawal, problem avoidance and wishful thinking are coping strategies that have been associated with the development of psychopathology including posttraumatic symptoms, anxiety and depression (e.g., Curran, Ponsford, & Crowe, 2000; Dougall et al., 2001; Griffing, 1998; Jeavons, de Horne, & Greenwood, 2000; Rainey, 1998; Stallard et al., 2001; Widows, Jacobsen, & Fields, 2000; Willert, 1996). It has been proposed that coping strategies that prevent adaptive emotional and information processing maintain posttraumatic symptoms (e.g., Clohessy & Ehlers, 1999). For example, avoidance, numbing and intrusive symptoms have been associated with escape-avoidance coping styles (e.g., Chung, Easthope, Chung, & Clark-Carter, 2001).

In contrast, problem engagement coping styles, such as taking positive actions to deal with exposure to a traumatic event, have been associated with less severe posttraumatic responses (e.g., Arambasic, Kerestes, Kuterovac-Jagodic, & Vizek-Vidovic, 2000). Use of cognitive restructuring and social support strategies have also been associated with positive posttraumatic adjustment (e.g., Shriner, 1999; Willert, 1996). The examination of the patterns of use of adaptive and maladaptive coping strategies has been proposed to identify targets for treatment interventions, and to focus on the reduced use of favoured maladaptive strategies, and increased use of adaptive strategies that may not be utilized to their full potential (e.g., Tobin & Griffing, 1995; Winje, 1998).

6.1.2 Personal beliefs and posttraumatic psychopathology

The propensity to cope in a particular way has been proposed to be mediated by underlying belief systems (e.g., Blanchard & Hickling, 1997). Psychological disturbance has been reported to result from irrational self-talk and

negative evaluation of life events. For example, Ellis (1962) theorized that it was not life events that caused psychological disturbance, but rather the individual's cognitive interpretation of the event that mediated affective and behavioural responses. Ellis formulated ten irrational beliefs, later generalized to three global beliefs (Kendall et al., 1995), that he described were commonly held by individuals exhibiting a wide range of psychological symptoms. Therefore, it may be speculated that the tendency to endorse a particular belief may pre-exist trauma exposure, and influence posttraumatic responses. In light of Ellis' theory, it was proposed that the nature of personal belief systems may enhance or hinder an individual's ability to cope with exposure to a traumatic event.

In terms of the association between personal beliefs and psychopathology, high positive correlations have been reported between severity of posttraumatic symptoms and dysfunctional beliefs concerning safety, invulnerability, trust, controllability of life events, self-esteem, and perceptions of the world as meaningful (e.g., Calhoun, Cann, Tedeschi, & McMillan, 1998; Ehlers et al., 1998; Foa & Riggs, 1993; Ho et al., 2000; Janoff-Bulman, 1992; Resick, Schnicke, & Markway, 1991; Suedfeld et al., 1998; Taylor, 1983; Wells et al., 2000; Wenninger & Ehlers, 1998). Dysfunctional beliefs have been proposed to operate as self-fulfilling prophecies in some individuals (e.g., McCann, Sakheim, & Abrahamson, 1988), suggesting that these beliefs result in maladaptive thoughts and behaviour and these ultimately negatively affect life experiences and outcomes. Trauma has been widely reported to challenge an individual's existing assumptions, beliefs and views about life and the world (e.g., Blanchard & Hickling, 1997; Bowman, 1999; Wheeler, 2001). Therefore, it has also been acknowledged that personal belief systems may be altered by traumatic experience (e.g., Arvay, 2001).

6.1.3 Aims and hypotheses

The aim of this study was to examine associations between coping styles and personal belief systems and the development of diagnostically distinct psychological responses to MVA trauma, and to introduce the characteristics of the sample used in this series of three integrated studies. As previously described, the use of maladaptive coping strategies and irrational belief systems have been associated with posttraumatic psychological distress in the literature. Therefore, it was hypothesized that:

1. Maladaptive coping strategies and irrational belief systems were expected to be associated with the PTSD group, and not the ASD or subclinical groups, as the latter groups have been proposed to reflect more adaptive posttraumatic psychological adjustment.
2. If coping styles and belief systems influenced the aetiology of ASD, differences in the ASD and subclinical profiles would be evident.

6.2 Method

6.2.1 Participants

Initial recruitment. Australian university students who had indicated their interest in participating in this series of studies by their response to the MVAQ (Holmes, 1997a) were contacted. Additional participants were recruited from the community in Tasmania by way of statewide radio, poster, pamphlet and newspaper advertising.

Exclusion criteria. The exclusion criteria for this series of studies were strict. Interested participants (N = 129) were screened by telephone in terms of

whether their MVA met the DSM-IV (APA, 1994) diagnostic criteria for a traumatic event, and only traumatic MVAs occurring between two months and ten years prior to the interview were included. It is acknowledged that a smaller window of time elapsed since trauma would have been desirable. However, with the limits of the population of Tasmania and the desired broad range of posttraumatic outcomes targeted by this project, it was not deemed possible to restrict inclusion criteria any further. The time frame was selected to facilitate the distinction between ASD and PTSD, and to maximize the accuracy of self-reported data, even though the affect laden nature of traumatic memories has been said to enhance accurate recall of events, including those occurring in excess of ten years prior to testing (e.g., Brewin, 1996; Winograd & Neisser, 1993). It was acknowledged that retrospective self-report of symptoms may be affected by time elapsed since trauma, and therefore diagnostic data were rigorously scrutinized using well recognized and utilized measures of lifetime psychopathology, a methodology supported by previous literature (e.g., Bolton, O'Ryan, Udwin, Boyle, & Yule, 2000; Grayson, Dobson, & Marshall, 1998; Keane et al., 1998; McFarlane, Bookless, & Air, 2001).

Following initial screening, 24 volunteers were excluded due to the experience of MVAs not meeting the criteria for a traumatic event, and a further four were excluded on the basis of time elapsed since the MVA. One hundred and one participants were interviewed following initial screening. A further 18 were excluded after interview due to self-reported history of non-MVA trauma (n = 10), and pre-MVA psychiatric diagnoses (n = 8). These exclusion criteria were selected in order to obtain diagnostic group comparisons with minimal interference from previous trauma exposure and psychiatric illness. Another

exclusion criterion was individuals who met the diagnosis for ASD and progressed to develop PTSD, as it was the intention in the present investigation to focus on the proposed subtype of ASD without progression to PTSD.

Selection of experimental groups. It was considered to include an additional two experimental groups representing the ASD to PTSD symptom course subtype and symptom-free responses, however, no volunteers met the criteria for these groups. It may be considered unusual that these response types were not found, given that previous reports (e.g., Bryant & Harvey, 2000a; Shalev, 1987) have highlighted these as more common outcomes. The symptom profiles and subsequent group membership will be presented in detail throughout this series of studies to provide evidence of the characteristics of the volunteer population.

The sample may have similarities to that described by Creamer and Manning (1998) in which none of the ASD group developed PTSD following an industrial accident. As reported by Bryant and Harvey (2000a), although ASD has been largely considered a precursor to PTSD, there is evidence to suggest that ASD may be a distinct entity predictive of longer term adjustment. As reported in chapter two, although high rates of progression from ASD to PTSD have been reported (e.g., Birmes et al., 2001, Holeva et al., 2002, Winston et al., 2002), this sample is representative of ASD leading to outcomes other than PTSD.

The final sample. The remaining sample included 83 participants (31 university students and 52 community volunteers). All participants were either drivers, passengers or pedestrians at the time of the accident. An information sheet and consent form were used to document informed consent for participation in the project (Appendix C-1). Participants were allocated to three groups on the basis of

DSM-IV criteria (PTSD, ASD and subclinical) in terms of the psychological sequelae experienced in response to the MVA. All ASD diagnoses were retrospective, in accordance with the aim of comparing the profiles of individuals who met the diagnostic criteria for ASD without progression to PTSD, with those individuals with PTSD and subclinical symptoms. There were no participants with responses meeting the criteria for PTSD with delayed onset.

This study described the full sample of participants used in this integrated series of three studies. Attempts were made to include the same sample of participants in all three studies to gain a comprehensive clinical picture of the psychological and psychophysiological correlates of PTSD, ASD and subclinical responses to MVA trauma. This aim was achieved in two of the three studies. The next study had a smaller sample, due to strict inclusion criteria with regard to memories of the MVA, as discussed in more detail in due course. Aside from the primary aim of conducting a multimodal analysis of individual responses, the design strategy of using the same participant group for multiple studies of a clinical issue has been demonstrated as a desirable approach because the statistical power of group data analysis may be more stable (e.g., Chassan, 1979), and participant attrition may be minimized (e.g., Kratchowill & Mace, 1984).

6.2.2 Materials

Structured clinical interview. At the time of assessment tool selection, the diagnostic category of ASD had only recently been introduced, and no published diagnostic measures were available to distinguish ASD from PTSD. Therefore, the Post-Accident Clinical Interview [PACI] (Holmes, 1997b) was designed for this study. Although structured interviews were available to assess PTSD, it was noted

that none were based on the DSM-IV criteria or specifically included ASD. Therefore, it was determined that the PACI would be designed to assess PTSD and ASD in accordance with the DSM-IV criteria, and that existing structured interviews, as reviewed in chapter four, would be used as guides in its development. Other researchers have faced a similar dilemma and have used the approach of developing their own instrument or modifying existing tools (e.g., Basoglu et al., 2001; Carlson et al., 2001; Vogel & Marshall, 2001).

The diagnostic component of the PACI (Holmes, 1997b) was strictly based on the DSM-IV criteria for PTSD and ASD (APA, 1994). Structured clinical interviews, particularly in the context of non-adversarial voluntary research, have been widely used in the current and retrospective (lifetime) diagnosis of anxiety disorders, and have been recommended to standardize data collection (e.g., Godart, Flament, Lecrubier, & Jeammet, 2000; Hickling & Blanchard, 1997; Regier, Rae, Narrow, Kaelber, & Schatzberg, 1998; Swendson et al., 1998). The PACI (Holmes, 1997b) was designed as a clinician-administered structured interview, and was used to elicit demographic, peritrauma and diagnostic information. The PACI (Holmes, 1997b) is displayed in Appendix C-2. The data collected using the PACI (Holmes, 1997b) was more recently coded against the CAPS for DSM-IV (Blake et al., 1998) and the ASDI (Bryant, Harvey, Dang, & Sackville, 1998), in order to address any concerns the reader may have in relation to accurate group allocation. Group membership was confirmed to be accurate using these comparisons. A case by case description of the lifetime posttraumatic symptom profile of each participant may be found in Appendix C-3. Current symptoms are further explored in chapter eight.

Coping styles. The Coping Strategies Inventory (CSI) (Tobin et al., 1984) was employed to assess adaptive and maladaptive coping styles participants used in dealing with MVA trauma. The CSI is a 72-item self-report scale that was based on the Ways of Coping Questionnaire (Folkman & Lazarus, 1980). The scale was designed to assess cognitive and behavioural coping strategies used to deal with a specific stressful event. In this study, participants were asked to consider how they coped with experiencing the MVA, and respond accordingly. Each item was rated on a five point Likert scale, reflecting the extent to which each coping strategy was used in dealing with the MVA. Each subscale consists of nine items, and each subscale score is the average of Likert ratings (1-5) on these items. The CSI is divided into eight coping style subscales as follows:

1. Problem solving: The cognitive and behavioural strategies employed to reduce stress by altering the problem situation.
2. Cognitive restructuring: The way in which the individual altered their interpretation of the problem situation using cognitive strategies, so that the event could be viewed in a more positive way.
3. Social support: The availability and use of emotional support.
4. Express emotions: The ability of the individual to release and express emotions.
5. Problem avoidance: The avoidance of cognitions or behaviours aimed at resolving the problem situation, including denial of the existence of the problem.
6. Wishful thinking: The use of cognitive strategies such as fantasising or hoping that the situation would improve, while demonstrating an inability or reluctance to use adaptive cognitive or behavioural strategies to deal with the problem.
7. Social withdrawal: Behavioural and emotional withdrawal from significant others as a result of the stressful event.

8. Self criticism: The extent to which the individual blamed and criticized him or herself for the event.

The eight subscales are categorised in terms of problem engagement/disengagement and problem/emotion focused coping strategies, as demonstrated in Table 8. The table also shows the Cronbach's alpha coefficients for the subscales. Problem engagement coping styles have been considered more adaptive than problem disengagement styles, and problem focused styles have been considered more adaptive than emotion focused styles (e.g., Tobin et al., 1984).

Table 8.

Categorisation and Cronbach's alpha coefficients of the eight subscales of the CSI.

	Problem engagement	Problem disengagement
Problem focused	Problem solving (.82)	Problem avoidance (.72)
	Cognitive restructuring (.83)	Wishful thinking (.78)
Emotion focused	Social support (.89)	Self criticism (.94)
	Express emotions (.89)	Social withdrawal (.81)

Test-retest coefficients, based on the context of the original stressor on retest, were reported by Tobin et al. (1984) as Problem solving .67, Cognitive restructuring .68; Social support .81; Express emotions .77; Problem avoidance .71; Wishful thinking .68; Social withdrawal .68; and Self criticism .83. The CSI (Tobin et al., 1984) is displayed in Appendix C-4.

Personal beliefs. The Beliefs Inventory (Davis, Eshelman, & McKay, 1995) was used to assess personal beliefs. The Beliefs Inventory is a modification

of the Irrational Beliefs Test designed by Jones (1968) that was based on Ellis' model of the 10 irrational beliefs proposed to be related to psychological maladjustment (Ellis & Harper, 1975). The Beliefs Inventory requires the respondent to agree or disagree with 100 statements of attitudes and beliefs about the world. The forced choice response differs from the wider range of response alternatives available in the Irrational Beliefs Test. The ten subscales derived from the Beliefs Inventory relate to the ten beliefs postulated by Ellis (Ellis & Harper, 1975). Endorsement of each belief is scored from one to ten, with higher scores indicative of greater endorsement of the belief. The belief subscales are:

B1 It is an absolute necessity for an adult to have love and approval from peers, family and friends.

B2 You must be unfailingly competent and almost perfect in all you undertake.

B3 Certain people are evil, wicked and villainous and should be punished.

B4 It is horrible when things are not the way you would like them to be.

B5 External events cause most human misery - people simply react as events trigger their emotions.

B6 You should feel fear or anxiety about anything that is unknown, uncertain or potentially dangerous.

B7 It is easier to avoid than face life's difficulties and responsibilities.

B8 You need something other or stronger or greater than yourself to rely on.

B9 The past has a lot to do with determining the present.

B10 Happiness can be achieved by inaction, passivity and endless leisure.

High subscale scores are related to irrationality and inflexibility of personal beliefs, with the maximum total score for the inventory score being 100. It has

been reported that irrational belief inventories have adequate reliability and face validity (e.g., Stebbins & Pakenham, 2001; Woodward, Carless, & Findlay, 2001). The Beliefs Inventory (Davis et al., 1995) is displayed in Appendix C-5.

6.2.3 Procedure

Ethical approval was obtained from the University of Tasmania Research Ethics Committee prior to participant recruitment for this series of three studies using the same sample. Voluntary participants were recruited from the university and community of Tasmania, as previously described. Structured clinical interviews (PACI; Holmes, 1997b) were then conducted, after which participants completed the CSI and the Beliefs Inventory. All interviews and assessments were conducted by the researcher who is a registered and practising clinical psychologist. As previously described, PACI (Holmes, 1997b) data were later coded against the CAPS for DSM-IV (Blake et al., 1998) and the ASDI (Bryant, Harvey, Dang, & Sackville, 1998) to scrutinize group membership.

6.2.4 Design and data analysis

A three group design was employed using the PTSD, ASD and subclinical group divisions based on posttraumatic diagnoses. Dependent variables were responses to the structured interview and self-report inventories. Structured interview data were presented using frequency and percentage conversions with one way ANOVA and chi square analyses. MANOVA, one way ANOVA with Bonferroni correction and Fisher Least Significant Difference (LSD) post hoc analyses were used to investigate between group differences in the psychometric scale scores. MANOVAs were employed due to their sensitivity not only to mean

differences, but also to the direction and size of correlations among the dependent variables. Significance levels were set at .05 for all tests, with Bonferroni corrections applied to reduce the possibility of Type 1 errors resulting from multiple significance tests. The Fisher LSD post hoc test for differences between pairs of means was adopted as it has been widely used following significant F tests (McPherson, 1990).

6.3 Results

6.3.1 Overview

The sample was described in terms of MVA details and demographics using data elicited by the structured interview (PACI; Holmes, 1997b). Responses to the CSI and the Beliefs Inventory were then compared between groups.

6.3.2 Participant demographics and MVA details

The participants in this study were 83 volunteers. Participant age ranged from 18 to 78 years (median age = 32), and there were no significant between group differences in age, F (2,80) = 2.75, $p > .05$, (PTSD M = 39.8, SD = 13.0; ASD M = 31.8, SD = 16.6; subclinical M = 31.9, SD = 14.7). Each group was comprised of more females than males, however, there were no significant differences in the ratio of females to males between groups, χ^2 (2, N = 83) = 1.00, $p > .05$. There were no significant between group differences in time elapsed since MVA, F (2,80) = 0.33, $p > .05$, (PTSD M = 87.6 months, SD = 92.7; ASD M = 73.8, SD = 155.7; subclinical M = 63.7, SD = 86.9), with time elapsed ranging from 2 to 98 months. In terms of posttraumatic diagnosis, 36% of the sample were

diagnosed with PTSD and 29% were diagnosed with ASD. Table 9 shows group frequencies and percentages of PACI (Holmes, 1997b) demographic and MVA items.

Table 9.

Group frequencies (and percentages) of PACI demographic and MVA detail item responses (N = 83).

PACI item	PTSD (n = 30)	ASD (n = 24)	Subclinical (n = 29)
	Freq.(%)	*Freq.*(%)	*Freq.*(%)
Demographic details			
Sex			
Female	22 (73)	18 (75)	18 (62)
Male	8 (27)	6 (25)	11 (38)
Marital status			
Single	13 (43)	11 (46)	13 (45)
Married/De facto	14 (47)	11 (46)	13 (45)
Other (e.g., Divorced/Widowed)	3 (10)	2 (8)	3 (10)
Education			
Secondary	7 (23)	6 (25)	7 (24)
Tertiary	23 (77)	18 (75)	22 (76)

(Table continues...)

Table 9 (continued...)

PACI item (Yes)	PTSD ($n = 30$)	ASD ($n = 24$)	Subclinical ($n = 29$)
	Freq.(%)	*Freq.*(%)	*Freq.*(%)
MVA details			
Role in MVA			
Driver	19 (63)	15 (62)	18 (62)
Passenger	10 (33)	8 (33)	10 (34)
Pedestrian	1 (3)	1 (4)	1 (3)
Trapped in MVA	10 (33)	10 (42)	8 (28)
Lost consciousness during MVA	8 (27)	6 (25)	8 (28)
Under influence of alcohol/drugs	1 (3)	1 (4)	1 (3)
Self injured in MVA	23 (77)	18 (75)	22 (76)
Hospitalized	17 (57)	13 (54)	16 (55)
Current post MVA physical pain	12 (40)	9 (37)	11 (38)
MVA litigation	12 (40)	10 (42)	12 (41)
Post MVA counselling	5 (17)	4 (17)	5 (17)
Prior MVA	18 (60)	14 (58)	17 (59)

There were no significant between group differences in marital status, χ^2 $(4, N = 83) = 2.14, p > .05$; education level, $\chi^2 (2, N = 83) = 2.76, p > .05$; role in MVA, $\chi^2 (4, N = 83) = 3.40, p > .05$; being trapped in the motor vehicle, χ^2 $(2, N = 83) = 3.33, p > .05$; losing consciousness in the MVA, χ^2 $(2, N = 83) = 5.74$, $p > .05$; reporting being under the influence of alcohol or other drugs at the time of

the MVA, χ^2 (2, N = 83) = 0.84, p > .05; being injured in the MVA, χ^2 (2, N = 83) = 2.00, p > .05, being hospitalized after the MVA, χ^2 (2, N = 83) = 2.42, p > .05, currently experiencing physical pain resulting from MVA injuries, χ^2 (2, N = 83) = 3.77, p > .05; involvement in post MVA litigation, χ^2 (2, N = 83) = 3.77, p > .05; receiving post MVA psychological counselling, χ^2 (2, N = 83) = 2.70, p > .05; or experiencing a less serious MVA prior to the traumatic MVA, χ^2 (2, N = 83) = 0.36, p > .05.

The group percentages showed that the majority of participants were either single or married/in a de facto relationship, tertiary educated, driving at the time of the MVA, injured in the MVA, hospitalized for treatment of physical injuries, and had experienced a less serious MVA than the traumatic MVA. A minority of the participants reported being trapped in the vehicle following the MVA, losing consciousness during the MVA, currently experiencing ongoing physical pain from injuries sustained during the MVA, and involvement in post MVA litigation. One participant in each group reported being under the influence of alcohol or other drugs at the time of the MVA. Only 17% of the participants reported receiving post MVA psychological counselling.

6.3.3 Coping strategies

MANOVA demonstrated that there were significant between group differences in mean scores on the CSI, *Rao's R* (16, 146) = 3.09, p = .0002. One way ANOVAs with Bonferroni correction were used to investigate the nature of these differences. Group means, standard deviations and one way ANOVA results on each subscale are shown in Table 10. The analyses demonstrated that the PTSD group rated use of Problem avoidance (*Fisher LSD* = 7.68, p < .05), Wishful

thinking (*Fisher LSD* = 7.45, $p < .05$), and Social withdrawal (*Fisher LSD* = 12.60, $p < .05$), more highly in dealing with the MVA than the ASD and subclinical groups. A trend was also found for the ASD group to rate Express emotions more highly than the subclinical group, however, this trend was not significant after Bonferroni correction. No other significant between group differences were found.

Table 10.

Group mean ratings (standard deviations) and one-way ANOVA results for each subscale of the CSI (N = 83).

Subscale	Group					
	PTSD	ASD	Subclinical	One way ANOVA		
	M (*SD*)	*M* (*SD*)	*M* (*SD*)	*F*	*p*	Post hoc
Problem solving	2.68 (2.22)	2.23 (0.71)	2.23 (0.92)	2.76	.07	na
Cognitive restructuring	2.90 (1.01)	2.76 (0.80)	2.49 (1.01)	1.39	.25	na
Express emotions	2.42 (0.84)	2.55 (0.77)	1.99 (0.84)	3.39	.04	A>S
Social support	2.51 (0.80)	2.96 (0.89)	2.58 (1.17)	1.60	.21	na
Problem avoidance	2.45 (0.74)	2.04 (0.55)	1.77 (0.56)	8.68	.0004*	P>A,S
Wishful thinking	3.16 (0.98)	2.62 (0.75)	2.21 (0.89)	8.46	.0004*	P>A,S
Self criticism	2.15 (1.14)	2.14 (1.24)	1.99 (1.26)	0.14	.86	na
Social withdrawal	2.81 (0.98)	1.87 (0.70)	1.72 (0.87)	13.69	.0001*	P>A,S

* Significant after Bonferroni correction (.05/8 = .006).

6.3.4 Personal beliefs

MANOVA demonstrated that there were no significant between group differences in mean scores on the Beliefs Inventory, *Rao's R* (20, 142) = 1.62, *p*

=.06. All groups achieved a moderate mean total score on the inventory (PTSD *M* = 46.73, *SD* = 13.31; ASD *M* = 44.58, *SD* = 14.04; subclinical *M* = 39.07, *SD* = 10.99). Group means and standard deviations on each scale are shown in Table 11.

Table 11.

Group means (standard deviations) for the Beliefs Inventory (N = 83).

Belief	Group					
	PTSD		ASD		Subclinical	
	M	*(SD)*	*M*	*(SD)*	*M*	*(SD)*
It is an absolute necessity for an adult to have love and approval from peers, family and friends.	3.60	(2.63)	3.91	(2.14)	4.14	(2.17)
You must be unfailingly competent and almost perfect in all you undertake.	4.03	(2.28)	4.00	(2.58)	3.31	(1.83)
Certain people are evil, wicked and villainous and should be punished.	4.90	(2.19)	5.25	(2.47)	4.38	(2.16)
It is horrible when things are not the way you would like them to be.	5.56	(1.90)	5.17	(1.86)	4.83	(2.35)
External events cause most human misery - people simply react as events trigger their emotions.	4.93	(2.50)	4.42	(2.26)	3.83	(2.24)
You should feel fear or anxiety about anything that is unknown, uncertain or potentially dangerous.	5.80	(2.92)	4.45	(3.05)	3.59	(2.53)
It is easier to avoid than face life's difficulties and responsibilities.	4.27	(1.89)	4.25	(1.85)	4.07	(2.20)
You need something other or stronger or greater than yourself to rely on.	4.60	(1.69)	5.50	(1.78)	4.48	(2.18)

(Table continues...)

Table 11 (continued...)

Belief	Group					
	PTSD		ASD		Subclinical	
	M	*(SD)*	*M*	*(SD)*	*M*	*(SD)*
The past has a lot to do with determining the present.	5.26	(2.41)	3.29	(2.27)	3.41	(2.13)
Happiness can be achieved by inaction, passivity and endless leisure.	3.77	(2.06)	4.25	(1.98)	3.45	(2.10)
Total score	46.73	(13.31)	44.58	(14.04)	39.07	(10.99)

* Significant after Bonferroni correction (.05/10 = .005).

6.4 Discussion

6.4.1 Participant demographics and MVA details

As predicted using the findings of the previous study, the sample comprised more females than males, however, each sex was equally represented in each of the experimental groups. The three groups were comparable in terms demographic characteristics, and represented a broad range of people in terms of age, marital status, education, accident details, and MVA outcomes. The participant characteristics were consistent with DIER's (2001) statistics regarding MVAs in Tasmania, suggesting that the sample was representative of the wider Tasmanian community. The characteristics of the participants were also comparable to the sample used for the Albany MVA Project (e.g., Blanchard & Hickling, 1997).

Interestingly, only 17% of the sample had participated in counselling after the MVA. It was not possible to determine from the data how many people were

offered and declined counselling, but the number who received counselling was small when the number of participants reporting posttraumatic symptoms was relatively high in this sample. In addition, the relatively low level of counselling may be considered noteworthy due to the fact that the majority of participants were female, and females have been reported to be more likely than males to engage in help-seeking behaviour after a traumatic event (e.g., Dobson, Grayson, Marshall, & O'Toole, 1998; O'Brien, 1998).

6.4.2 Coping styles and personal beliefs

Maladaptive coping strategies and irrational belief systems have been associated with posttraumatic psychological distress in the literature (e.g., Curran et al., 2000; Dougall et al., 2001; Griffing, 1998; Jeavons et al., 2000; Rainey, 1998; Stallard et al., 2001; Widows et al., 2000; Willert, 1996). The hypothesis that these features were expected to be associated with the PTSD group, and not the ASD or subclinical groups, was partially supported.

The use of maladaptive coping strategies was found to be associated with PTSD, in comparison to the ASD and subclinical groups. The use of problem avoidance, wishful thinking and social withdrawal strategies were rated more highly by the PTSD group in dealing with traumatic MVAs, and these findings were consistent with previous literature (e.g., Curran et al., 2000; Dougall et al., 2001; Griffing, 1998; Jeavons et al., 2000; Rainey, 1998; Stallard et al., 2001; Widows et al., 2000; Willert, 1996).

Problem avoidance as a coping strategy is particularly problematic following exposure to trauma. Avoidance of trauma-related reminders is symptomatic of PTSD (APA, 1994) and exposure therapy for PTSD has been

developed to counter the negative effects of avoidance and other posttrauma symptomatology (e.g., Foa, Molnar, & Cashman, 1995; Frueh, Turner, & Beidel, 1995; Paunovic, 1997; Richards & Rose, 1991; Thompson, Charlton, Kerry, & Lee, 1995). A propensity to avoid facing problem situations may exacerbate avoidance symptomatology or prolong the duration of symptoms.

In addition, the adoption of social withdrawal as a strategy for managing the psychological effects of an MVA by people with PTSD could exacerbate posttrauma symptomatology. It has clearly been demonstrated that low levels of social support have been related to more severe PTSD symptomatology (Eriksson, 1997; Litzinger, 1997), and that accessing social support networks moderates PTSD symptom levels (e.g., Fontana, Rosenheck, & Horvath, 1997). Therefore, actively withdrawing from social networks would be likely to be self-defeating and increase posttrauma symptomatology in the longer term.

Interestingly, there were no significant between group differences in the reported use of more adaptive coping strategies such as problem solving, cognitive restructuring, expressing emotions and utilizing social support, or the maladaptive coping strategy of self-criticism. This finding suggests that the PTSD group did not report lack of adaptive coping skills, but rather the utilization of multiple adaptive and maladaptive strategies.

Groups with other types of significant psychopathology have been demonstrated to have combinations of both adaptive and maladaptive coping strategies available to them (Watson, 1997) as have people with health-related problems (Hancock, Craig, Tennant, & Chang, 1993; Jahanshahi, 1991). Research in other areas has indicated that people who negatively evaluate self and others were less likely to access adaptive coping strategies such as task-orientation and

support-seeking behaviour and were more likely to use maladaptive coping strategies such as escape-avoidance styles of coping (Rothbard, 1997). Interestingly, when considering chronic pain populations, it has been demonstrated that it was only maladaptive coping strategies that contributed to the prediction of self-perceived disability and not the use of adaptive coping strategies (Bowling, 1996). It may be that when overwhelmed by events, the ability to adequately use adaptive coping strategies and suppress the use of maladaptive coping strategies is lost. This view is supported by research that has indicated that people with a strongly positive view of life difficulties were better able to maximise adaptive coping and reduce reliance on maladaptive coping strategies, and reduce distress as a consequence, whereas people who held negative views about life were not able to do so (Stoddard, 1995).

It was also hypothesized that if coping styles and belief systems influenced the aetiology of ASD, differences in the ASD and subclinical profiles would be evident. No significant differences were found in the ASD and subclinical profiles, suggesting that coping styles and belief systems were not mediating factors in the differential development of ASD and subclinical responses. The only trend for a difference between the coping style profiles of the ASD and subclinical groups was for the ASD group to rate the use of expressing emotions more highly. As previously reported, the difference was not significant after Bonferroni correction, but may warrant further exploration.

The reported use of each strategy by the groups was moderate, with the most endorsed strategy for each group being wishful thinking for the PTSD group, and social support for the ASD and subclinical groups. These results are consistent with the literature that reported maladaptive coping strategies may be associated

with severity of posttraumatic symptoms, and adaptive coping strategies such as utilization of social support may reduce, or mediate the development of, posttraumatic symptoms (e.g., Blanchard, Hickling, Taylor, & Loos, 1995; Falsetti, 1997; Fontana & Rosenheck, 1998; Griffing, 1998; Hovanitz & Kozora, 1989; Livneh et al., 2000; Rainey, 1998; Shriner, 1999; Tunnecliffe & Tunnecliffe, 1997; van der Kolk, McFarlane, et al., 1996; Willert, 1996).

As proposed by Tobin and Griffing (1995), it may be beneficial in the treatment of PTSD to target the use of maladaptive coping strategies, and encourage the increased use of adaptive strategies. The results indicate that the PTSD group endorsed the use of adaptive strategies, and thus were not devoid of these skills, providing a rationale for transfer in strategy use, as opposed to learning new skills. It is proposed in light of these results that early assessment of coping styles may also serve as a prognostic indicator, given the diagnosis-specific profiles produced by the findings.

As previously indicated, it was also hypothesized that the PTSD group would report more inflexible and irrational belief systems than the other groups, as found with other trauma types and populations (e.g., Calhoun et al., 1998; Ehlers et al., 1998; Foa & Riggs, 1993; Ho et al., 2000; Janoff-Bulman, 1992; Resick et al., 1991; Suedfeld et al., 1998; Taylor, 1983; Wells et al., 2000; Wenninger & Ehlers, 1998). This was found not to be the case. There were no significant between group differences in response to the Beliefs Inventory. The total group scores were moderate, and reflected that all groups reported flexible and rational belief systems. Thus, there was little evidence found to suggest that belief systems influenced the development of posttraumatic stress disorders. It is interesting to note that PTSD and ASD did develop without irrational belief systems. It was

noted by van der Kolk and McFarlane (1996) that belief systems may have most influence on psychological response when the traumatic event is considerably out of the range of everyday experience. Further, due to the prevalence of MVAs in the community, it may be that responses to MVAs are less likely than rare events to be affected by personal belief systems. The results suggest that the three groups, to some extent, were able to adjust to the experience of MVA trauma, by flexibly integrating the experience into a realistic and rational view of the world.

In relation to coping styles and personal beliefs, it was acknowledged that these variables may be affected by the experience of a traumatic event, but that the propensity to cope or view the world in a particular way, may pre-exist the trauma. A cross-sectional design with posttraumatic measures of these variables cannot ascertain the degree to which the presented profiles were influenced, if at all, by trauma exposure.

6.4.3 Summary and conclusions

The results indicate that the aetiologies of PTSD and ASD are associated with different coping style profiles, but not belief systems. No significant differences were found in the coping and belief profiles of the ASD and subclinical groups to explain the aetiologies of these distinct posttraumatic pathways. The distinction between the coping profiles of the PTSD and ASD groups may provide information about the role of coping mechanisms in more adaptive recovery from posttraumatic symptoms associated with the ASD diagnosis. That is, the adaptive coping profile of the ASD group, with significantly less use of maladaptive coping strategies than the PTSD group, may highlight a mechanism by which the ASD group avoided the development of PTSD.

The coping style profiles presented in this study may be considered to identify potential treatment targets and prognostic indicators. The profiles support previous findings of the importance of coping mechanisms as moderators of life event stress-psychopathology relationships (e.g., Blanchard & Hickling, 1997; Hovanitz & Kozora, 1989; van der Kolk, McFarlane, et al., 1996). However, as highlighted by the proposed integrated aetiological theory of posttraumatic stress disorders, it is not only coping mechanisms that may play a role in different posttraumatic adjustment pathways. The next study shifts the focus from coping styles and personal beliefs to the recollection of responses occurring at the time of trauma exposure, in this multimodal investigation of the development of diagnostically distinct posttraumatic responses to MVA trauma.

This study has been previously presented in part, and referenced contributions by the author of this thesis are underlined:

Holmes, G.E., Williams, C.L., & Haines, J. (1998a, August). *Cognitive correlates of posttraumatic responses to motor vehicle accidents.* Paper presented at the 56th Annual Convention of the International Council of Psychologists, Melbourne, Australia.

Holmes, G.E., Williams, C.L., & Haines, J. (2001a). Cognitive profiles of Acute Stress Disorder and Posttraumatic Stress Disorder following motor vehicle accident trauma. In R. Roth & S. Neil (Eds.), *A matter of life: Psychological theory, research and practice* (pp.277-285). Lengerich, Germany: Pabst Science. (Appendix C-6)

CHAPTER SEVEN

STUDY THREE:

PSYCHOPHYSIOLOGICAL AND PSYCHOLOGICAL RESPONSES TO RECALL OF TRAUMATIC AND NON-TRAUMATIC MEMORIES

7.1 Introduction

Peritraumatic responses have been considered to present particularly difficult challenges in terms of measurement and assessment (e.g., Williston, 2001). The logistical constraints of targeting responses that occur at psychophysiological and psychological levels during the course of a traumatic event have restricted practical approaches to eliciting this information. One method of accessing psychophysiological and psychological reactivity to peritraumatic cues has been the use of idiosyncratic imagery (e.g., Blanchard & Hickling, 1997; Carson et al., 2000; Pitman et al., 2001). This tool has been deemed useful in the analysis of psychophysiological and psychological reactivity to memories of problematic events (e.g., Brain, Haines, & Williams, 1998; Haines, Josephs, Williams, & Wells, 1998; McLaren, Haines, & Williams, 1996; Williams, Wilson, Montgomery, & Batik, 1989). An adaptation of the use of guided imagery was employed in this study, and was proposed to extend knowledge of reactivity to trauma-related cues provided by other studies and reviews (e.g., Blanchard & Hickling, 1997; Carson et al., 2000; Grey, Holmes, & Brewin, 2001; Keane et al., 1998; Pitman et al., 2001; Williston, 2001; Wolfe et al., 2000).

7.1.1 Psychophysiological assessment of trauma responses

The investigation of the nature of trauma responses through their expression at the psychophysiological level (e.g., Southwick, Bremner, Krystal, & Charney, 1994) has resulted in the development of multimodal psychophysiological assessment tools, as previously discussed. McFarlane (1999) suggested that the relationship between biological and psychological responses to trauma may be

considered central to understanding the development of PTSD, and that critical neurobiological changes may occur at the time of the trauma which moderate the magnitude and type of trauma response. He proposed that the central underlying question may be considered the extent to which peritraumatic psychophysiological and psychological reactions are related to the psychopathology and neurobiology of any disorder that emerges.

7.1.2 Multimodal assessment of PTSD

Physiological reactivity to trauma-related cues has been included as one of the diagnostic criteria for PTSD (APA, 1994) and has been proposed as a behavioural expression of underlying biological processes (e.g., Glod & McEnany, 1995; Shalev, 1999). Indeed, Blanchard, Hickling, Vollmer, and colleague (1995) suggested that hyperarousal symptoms were more enduring than other posttraumatic symptoms. The DSM-IV (p. 426, APA, 1994) has recommended the measurement of increased arousal associated with PTSD by studying autonomic functioning via measures such as heart rate, electromyography (EMG) and electrodermal responses.

As discussed in chapter four, various stimuli have been used to elicit psychophysiological responses which may be used to distinguish PTSD from non-PTSD responses to trauma. These stimuli have included audiotaped trauma cues, visual presentation of trauma-related images, the presentation of standardized odours, and virtual reality simulations of trauma-related environments (e.g., Blanchard, Kolb, Gerardi, Ryan, & Pallmeyer, 1986; Carlson, Singelis, & Chemtob, 1997; Hyer, Arena, O'Leary, & Elkins, 1985; Keane et al., 1998; Kolb, 1987; Liberzon, Taylor, et al., 1999; Litz, Orsillo, Kaloupek, & Weathers, 2000;

Malloy, Fairbank, & Keane, 1983; McCaffrey et al., 1993). Results have indicated across trauma types that although heightened responsivity to trauma-related cues was apparent in relation to the experiences of individuals diagnosed with PTSD in comparison to non-PTSD groups, the response was not always evident in all modalities (e.g., Blanchard & Buckley, 1999; Pallmeyer, Blanchard & Kolb, 1986).

To counter issues relating to stimulus-response specificity and individual response stereotypy it was suggested that psychophysiological investigations require multiple measures to comprehensively assess patterns of response within and between experimental groups (e.g., Blanchard & Buckley, 1999; Blanchard et al., 1991; Stern, Ray, & Davis, 1980). Heart rate and peripheral blood flow measures have been reported to be the strongest modes of measuring psychophysiological reactivity to trauma-related stimuli, and facilitating discrimination between PTSD and non-PTSD trauma responses (e.g., Blanchard et al., 1991; Blanchard et al., 1999).

Other variables have been advocated due to the specific activities they measure. For example, EMG has been used to examine tension within muscles, respiration has been used to examine the supply of oxygen to cells and the removal of carbon dioxide, and skin conductance level (SCL) has been selected to assess electrodermal activity (e.g., Carson et al., 2000; Pitman et al., 2001; Rothbaum et al., 2001; Stern et al., 1980; Wolfe et al., 2000). A recent review of psychophysiological assessment findings in PTSD concluded that psychophysiological assessment has valuable clinical applications, including diagnostic and treatment utilities (e.g., Orr & Roth, 2000).

7.1.3 The psychophysiology of ASD

Finding an objective measure of ASD is arguably a more difficult task because, by definition, the disorder is not associated with long term psychophysiological or biological change (APA, 1994). However, it was proposed that there may be objective indicators reflected in peritraumatic psychophysiological processes, which, if they could be accessed, could identify prognostic features of ASD as distinct from those of PTSD and subclinical responses. For example, the emphasis on peritraumatic dissociative phenomena in ASD criteria (APA, 1994) may suggest that distinct psychophysiological mechanisms occur at the time of trauma exposure in individuals who develop this disorder. It was proposed that if trauma-related cues could stimulate reexperiencing of peritraumatic responses, then processes such as peritraumatic dissociative phenomena may be assessed.

7.1.4 Peritraumatic psychological responses

The DSM-IV (APA, 1994) criteria for the definition of a traumatic event reflect that psychological responses experienced at the time of the trauma may be considered influential in the development of PTSD and ASD. Peritraumatic emotional reactions predictive of the later development of posttraumatic symptoms have been reported to include fear, numbing, guilt and anger (e.g., Bernat, Ronfeldt, Calhoun, & Arias, 1998; Foy, Osato, Houskamp & Neumann, 1992; Radnitz et al., 1998; Roemer, Orsillo, Borkovec, & Litz, 1998; Sivik, Delimar, Korenjak, & Delimar, 1997). It is suggested that the examination of patterns of psychological responses to peritraumatic and neutral cues may identify psychological factors in the development of differential posttraumatic responses.

Indeed, it has been noted that the emotional processes associated with PTSD and ASD have been the least understood and the most understudied aspect of the disorders (Litz et al., 2000).

Peritraumatic factors such as perceptions of injury severity to self and others (e.g., Blanchard & Hickling, 1999; Blanchard, Hickling, Mitnick et al., 1995) and fear of death (e.g., Buckley, 2000; Foa, Steketee & Rothbaum, 1989; Ullman & Filipas, 2001) have been associated with the severity of posttraumatic symptoms. Peritraumatic environmental factors such as being trapped in the vehicle, being physically injured and being involved in a single vehicle MVA have been associated with the experience of a peritraumatic death imprint (PDI), a specific type of fear of death. A PDI has been defined by Haines, Williams, Holmes, and colleague (2000) as a cognitive experience that occurs during the course of a traumatic event that is characterized by an individual believing that their own death is imminent. No age or sex differences have been found in the prevalence of a PDI in MVA survivors (Haines, Williams, Holmes, et al., 2000; Haines, Williams, Holmes, Wells, et al., 2001; Haines, Williams, Mycak, et al., 2000).

It has been proposed that the examination of psychological responses in isolation may limit the understanding of posttraumatic psychological mechanisms. It has been suggested that the integrated examination of subjectively reported psychological responses in combination with objectively measured psychophysiological responses may provide a more comprehensive multimodal analysis of posttraumatic responses (e.g., Griffin, Resick, & Mechanic, 1997).

7.1.5 Guided imagery and psychophysiological assessment

Guided imagery is a tool that has been used in the assessment, diagnosis and treatment of PTSD (e.g., Boudewyns & Hyer, 1990; Carson et al., 2000; Fidaleo, Proano, & Friedberg, 1999; Pitman et al., 1987; Shin et al., 1999). Previous studies using guided imagery to elicit arousal responses to personalized trauma cues (e.g., Blanchard & Hickling, 1997; Boudewyns & Hyer, 1990; Orr, Pitman, Lasko, & Herz, 1993; Pitman et al., 1987; Pitman & Orr, 1990; Shalev, Orr & Pitman, 1992) have contributed much to the body of knowledge of posttraumatic responses. These studies have supported the proposition that PTSD and non-PTSD responses to trauma may be differentiated on the basis of multiple psychophysiological variables. It was proposed that the findings of these studies may be meaningfully extended by the examination of the nature and patterns of emotional response integrated with associated psychophysiological response patterns. In addition, these patterns may be examined over time, across the stages of an event. It was proposed that the analysis of response patterns, occurring throughout the longitudinal course of an event, may assist in the discrimination of PTSD, ASD and subclinical responses to trauma.

The four stage guided imagery methodology has been described as a structured clinical tool which incorporates the use of cognitive, behavioural, environmental, emotional and psychophysiological information in the objective measurement of individual responses to real life events (e.g., Brain, Haines, Williams, Stops, & Driscoll, 1996). The methodology has been used to examine life events and psychiatric disorders including self-mutilation (e.g., Brain et al., 1998; Brain, Williams, & Haines, 1996; Haines, Williams, Brain, & Wilson, 1995), self-poisoning (e.g., Driscoll, Williams, & Haines, 1996; Williams, Haines, Lester,

& Rooke, 2001), police stress (e.g., McLaren et al., 1996), domestic violence (e.g., Williams et al., 1989), Obsessive-Compulsive Disorder (e.g., Haines et al., 1998), workplace phobia (e.g., Carson, Haines, & Williams, 1998); Bulimia Nervosa (e.g., Williams, Haines, & Brain, 1995), Dissociative Identity Disorder (e.g., Williams, Haines, & Sale, 2002) and homicidal behaviour (e.g., Glading, Williams, & Haines, 2001; Haines, Williams, Sale, Glading, & Davidson, 2002; Williams, Haines, & Casey, 2000).

The tool was originally conceived for forensic use in the examination of an act of filicide (*R. v. Horton*, 1986). Guided imagery describing punitive interactions between a mother and her child was used in the successful defence of the young mother. The methodology used personalized scripts, each divided into four chronological event stages, to compare reactivity to specific elements of actual events experienced by the participant. Unlike other guided imagery methodologies, this methodology facilitated the examination of patterns of reactivity throughout the course of recall of specific events and behaviour. This methodology should not be confused with the methodology used by Keane and colleagues (1998) that used four thirty second time periods to compare responses to baseline rest, stimulus reading, recall and post recall recovery. The four stage guided imagery methodology refers exclusively to measuring responses to the recall of one event in four, idiosyncratic, chronological stages. The methodology was designed to compare concurrent emotional and arousal states in response to specific stimuli.

The tool has been employed to access and measure responses to recall of real life events, as proposed by the bio-informational theory of emotional imagery (Lang, 1979). Studies using the methodology have advocated the auditory mode of verbally presented idiosyncratic stimuli, which is a methodology endorsed by other

researchers (e.g., Blanchard & Buckley, 1999). Previous studies utilizing this methodology in the examination of other psychiatric disorders in retrospective and current states of diagnosis have demonstrated that recovery does not affect psychophysiological patterns to imagery of past events (Brain et al., 1998). However, with respect to MVA survivors, Blanchard and colleagues (1996) found that time elapsed since trauma exposure did influence psychophysiological responses. It is therefore acknowledged that measures of recall of traumatic memories may not mirror the exact responses occurring at the time of trauma exposure due to influences such as learning from the observations of others, and being made aware of additional details that may shape responses to memories of the event.

Posttraumatic response patterns may, however, provide longer term indicators of the manifestation of posttraumatic response types. As stated by Blanchard and Hickling (1997), psychophysiological studies focusing on recent trauma have largely replicated findings of psychophysiological studies using participants with decades elapsed since trauma exposure, such as the Vietnam veteran population. It was proposed that the application of the four stage guided imagery tool to posttraumatic responses following MVA trauma may provide an objective clinical assessment methodology to distinguish PTSD, ASD and subclinical responses by the analysis of integrated psychological and psychophysiological response patterns.

7.1.6 The MVA and the post accident scene

The four stage guided imagery methodology has been reported to facilitate analysis of psychophysiological responses occurring during the course of recall of

specific events, allowing response patterns to be observed for distinguishing features (e.g., Haines et al., 1995). It was proposed that this utility may be extended, not only in terms of the comparison of differential posttraumatic diagnoses and MVA trauma, but in the use of peri and post event scripts. This extension of the methodology was theorized to target and compare patterns and intensity of psychophysiological responses to the MVA and post accident events occurring at the trauma scene, facilitating a logical progression from previous work in the area (e.g., Blanchard & Hickling, 1997).

Considerable discussion has occurred regarding factors contributing to the development and maintenance of posttraumatic stress disorders. Periaccident factors such as experiencing a PDI (Haines, Williams, Holmes, et al., 2000; Haines, Williams, Holmes, Wells, et al., 2001) and incidents occurring at the accident scene following the MVA, such as distressing interactions between drivers, have been proposed as possible contributing factors to negative long term responses (e.g., Blanchard & Hickling, 1997). Anecdotally, individuals who have experienced an MVA sometimes report that events occurring at the scene of the accident are more traumatic than the accident itself. For example, observing the aftermath of the accident including seeing bodies and blood, may be more traumatic than experiencing the physical impact of the MVA. It was proposed that the identification of the point at which the most intense distress occurs during the recall of an MVA and its immediate aftermath may provide valuable intervention and treatment targets. Between group differences may indicate elements of the trauma that are predictive of the development of PTSD and ASD, thus facilitating early assistance for vulnerable individuals, which may circumvent chronic responses.

7.1.7 Aims and hypotheses

This study proposed a method of exploring the relationship between biological and psychological responses to trauma, and the subsequent development of distinct posttraumatic responses. A four stage guided imagery methodology was used to investigate psychological and psychophysiological response patterns throughout the course of recall of a traumatic event, and nontraumatic events for comparative purposes. Two non traumatic events were used in order to control for the effects of low versus high arousal nontraumatic cues. It was proposed that the use of the four stage guided imagery methodology would provide information that, when integrated with existing research, would clarify the role of peritraumatic psychophysiological and psychological responses in the differential development of posttraumatic stress disorders. Patterns of response were compared between the PTSD, ASD and subclinical groups; imagery script types; and script stages. It was hypothesized that:

1. Imagery ability would not differ between groups, reflecting comparable skills in performing the experimental tasks.

2. Personalized traumatic imagery would provoke greater psychological and psychophysiological reactivity than personalized nontraumatic imagery for all groups and that each group would demonstrate a diagnostically distinct pattern of reactivity in response to traumatic imagery.

3. The psychological and psychophysiological response of the ASD group would reflect evidence of recall of peritraumatic dissociative phenomena.

4. Psychological states measured by visual analogue scales (VASs) would reflect group specific patterns of response to posttraumatic cues.

5. The experience of a PDI would be associated with severity of posttraumatic symptoms and, therefore, be most frequently reported by the PTSD group.

It was proposed that the characteristics of between group differences on the various measures would provide further evidence of PTSD and ASD as distinct diagnostic entities, and would identify further information about the mechanisms underlying the development of these disorders. The study aimed to build on the foundation of the existing body of knowledge of the psychology and psychophysiology of trauma responses for the purpose of refining the assessment, diagnosis and treatment of posttraumatic stress disorders.

7.2 Method

7.2.1 Participants

Participants were 51 individuals who had been exposed to an MVA meeting the DSM-IV definition of a traumatic event (APA, 1994), and were selected from the larger sample participating in studies two and four. The sample included 19 individuals diagnosed with PTSD, 15 individuals diagnosed with ASD without progression to PTSD, and 17 individuals with subclinical responses to MVA trauma. Specific symptom profiles of these participants are referred to in Appendix C-3. Each group included seven males. Exclusion criteria, in addition to those noted in the previous study, were substance use potentially affecting psychophysiological responses ($n = 8$), and loss of consciousness during the MVA ($n = 24$).

Participant age ranged from 18 to 78 years, and there were no significant between group differences in age, $F(2,48) = 2.89$, $p > .05$, (PTSD $M = 38.7$ years,

SD = 12.7; ASD *M* = 32.3, *SD* = 17.2; subclinical *M* = 32.1, *SD* = 15.2). Each group was comprised of more females than males, however, there were no significant differences in the ratio of females to males between groups, χ^2 (2, *N* = 51) = 1.00, $p > .05$. There were no significant between group differences in time elapsed since MVA, $F(2,48) = 0.34$, $p > .05$, (PTSD *M* = 85.6 months, *SD* = 91.7; ASD *M* = 72.8, *SD* = 142.7; subclinical *M* = 65.7, *SD* = 83.9), and time elapsed ranged from 2 to 98 months. Table 12 displays the participants' characteristics in detail.

Table 12.

Participant characteristics by frequency (% of group) (N = 51).

PACI item	PTSD (*n* = 19)	ASD (*n* = 15)	Subclinical (*n* = 17)
	Freq. (%)	*Freq.* (%)	*Freq.* (%)
Sex			
Female	14 (74)	11 (73)	12 (71)
Male	5 (26)	4 (27)	5 (29)
Marital status			
Without partner	10 (53)	8 (53)	9 (53)
Married/De facto	9 (47)	7 (47)	8 (47)
Education			
Secondary	5 (29)	4 (27)	5 (26)
Tertiary	14 (71)	11 (73)	12 (74)

(Table continues...)

Table 12 (continued...)

PACI item	PTSD (n = 19)	ASD (n = 15)	Subclinical (n = 17)
	Freq. (%)	*Freq.* (%)	*Freq.* (%)
Role in MVA			
Driver	11 (58)	9 (60)	11 (65)
Passenger	7 (37)	5 (33)	5 (29)
Pedestrian	1 (5)	1 (7)	1 (6)
Trapped in MVA (Yes)	5 (26)	5 (33)	4 (24)
Self injured in MVA (Yes)	14 (74)	12 (80)	12 (71)
Current post MVA pain (Yes)	7 (37)	4 (27)	4 (24)
MVA litigation (Yes)	7 (37)	5 (33)	5 (29)
Post MVA counselling (Yes)	5 (17)	4 (17)	5 (17)

There were no significant between group differences in marital status, χ^2 $(2, N = 51) = 2.15, p > .05$; education level, χ^2 $(2, N = 51) = 2.76, p > .05$; role in MVA, $\chi^2 (4, N = 51) = 3.40, p > .05$; being trapped in the MVA, $\chi^2 (2, N = 51) = 3.34, p > .05$; being injured in the MVA, χ^2 $(2, N = 51) = 2.00, p > .05$, currently experiencing physical pain resulting from MVA injuries, χ^2 $(2, N = 51) = 3.67, p > .05$; involvement in post MVA litigation, χ^2 $(2, N = 51) = 3.67, p > .05$; or receiving post MVA psychological counselling, $\chi^2 (2, N = 51) = 2.68, p > .05$

The group percentages showed that the majority of participants were without partner, tertiary educated, driving at the time of the MVA, and injured in the MVA. Fewer than half of the participants reported being trapped in the vehicle following the MVA, currently experiencing ongoing physical pain from injuries

sustained during the MVA, involvement in post MVA litigation, and receiving post MVA psychological counselling.

7.2.2 Materials

7.2.2.1 Scales

Imagery ability. The Betts QMI Vividness of Imagery Scale (Sheehan, 1967) was used to assess imagery ability. This scale was designed to measure the ability to form vivid mental images of standard stimuli in seven stimulus modalities (visual, auditory, gustatory, olfactory, kinesthetic, cutaneous and organic). This scale has been used as a measure of imagery ability in previous research (e.g., Laor et al., 1999).

Psychological responses to imagery. VASs (e.g., Ahles, Ruckdeschel, & Blanchard, 1984; McCormack, de Horne, & Sheather, 1988; Stern, 2000) were used to assess psychological responses to imagery. VAS scores (from 0 to 100) represented psychological responses on the following dimensions relating to responses: relaxed/tense, calm/angry, happy/sad, not guilty/guilty, normal/unreal, normal/numb, unafraid/afraid, and comfortable/uncomfortable. These dimensions were selected on the basis of psychological states commonly associated with trauma, as reflected by the DSM-IV criteria for PTSD and ASD (APA, 1994), including tension, anger, sadness, guilt, fear and discomfort, and the feelings associated with dissociative phenomena, unreality and numbness. Higher scores were indicative of a more negative experience. In addition, VASs were also used as control measures to assess the experience of guided imagery including the clarity of imagery (unclear/clear), the accuracy of script content and relation of the content to real life experiences (not close/very close), and the ability to concentrate on

imaging the scene (very distracted/very well). High scores on these scales were reflective of clear imaging, accurate script content, and freedom from distractibility during the imaging tasks. All scales were rated for each of the four stages of each script. VASs were chosen for this task because they have been reported to place minimal cognitive demands on the respondent and they have been specifically designed to assess internal mood states (Stern, 2000). Copies of VASs utilized in this study are included in Appendix D-1.

7.2.2.2 Imagery scripts

All participants were interviewed to collect information for four personalized guided imagery scripts. The scripts were:

1. Low arousal neutral (LAN): A description of an emotionally neutral event associated with low psychophysiological arousal (e.g., making a cup of tea).
2. High arousal neutral (HAN): A description of a nontraumatic event associated with high psychophysiological arousal (e.g., routine exercise).
3. MVA: A description of the MVA from the moments before the impact until the moments immediately following the impact.
4. Post MVA: A description of the events occurring at the scene of the MVA from the moments immediately following the impact until leaving the accident scene.

Four separate scripts detailing personalized event descriptions were written for each participant. Scripts were divided into four stages: setting the scene (stage one), approach (stage two), incident (stage three) and consequence (stage four). Each stage contained personalized multi-sensory descriptions containing both stimulus and response content, providing specific cues of recollections of the

environment, behaviours, thoughts, feelings, and psychophysiological reactions experienced during the script event. The inclusion of stimulus and response information in personalized imagery content has been endorsed by other researchers in this field (e.g., Blanchard & Buckley, 1999), because this combination of cues has been proposed to provide the most detailed recall of life experiences. Stage one (setting the scene) described the environment in which the event took place and the context of the situation, stage two (approach) described the immediate lead up to the event, stage three (incident) described the actual event taking place, and stage four (consequence) described reactions and immediate actions taken in response to the event. Imagery scripts contained only those elements recalled by the individual participant, using the language expressed by the participant. Examples of each script type are presented in Appendix D-2.

7.2.2.3 Apparatus

Psychophysiological responses were recorded using a Power Macintosh 7300/180 computer linked to a MacLab/8S data acquisition system using Chart version 3.5.6 software. Recordings were made at 1mm/s-1 with a sampling frequency of 200 samples/s-1. Finger blood volume (FBV) was measured using a photoelectric plethysmograph fitted to the distal phalange of the second finger of the non-dominant hand using a Velcro fastener. The Plethysmograph was connected through a GP Amp coupler with the amplifier range set at 10V and a band-pass filter setting of DC to 10Hz. The electrocardiograph (ECG) was measured using 7mm Ag/AgCl electrodes fitted on both sides of the torso at the level of second rib with an earth references on the left mastoid process. The electrodes were input through a BioAmp coupler, amplifier range used was 1 mV

full scale and the band-pass filter setting was 0.3 to 50Hz. The ECG signal was input to a second channel and converted to beat-to-beat heart rate (HR), and the range setting was 0 to 200 bpm. Respiration (RESP) was monitored using a Pneumotrace respiration transducer belt placed under the arms and around the chest. SCL was measured using 10mm Ag/AgCl electrodes on the first and third fingers of the participant's non-dominant hand. EMG was monitored by two Ag/AgCl adhesive electrodes placed 1/3 and 2/3 above the supraorbital margin. A range of psychophysiological measures were selected to account for stimulus-response specificity and individual response stereotypy (Fleming & Baum, 1987; Stern et al., 1980).

7.2.3 Procedure

Participants were asked to describe the traumatic MVA and two neutral events of their choice. This information was recorded on audio-tape. The tape was used by the researcher after the session to produce personalized guided imagery scripts, using only the information provided by each participant on the tape, and using the actual words and language style used by the participant in their description. Participants were asked at the conclusion of the first session to complete the Betts QMI Vividness of Imagery Scale (Sheehan, 1967).

At the commencement of the second session, electrodes were fitted. Participants were asked to sit in a comfortable chair while psychophysiological measures were calibrated. The researcher remained in the room with the participant while a second experimenter monitored psychophysiological responses on the computer outside the room. Communication was achieved via intercom. Psychophysiological measures were taken as the four personalized guided imagery

scripts were read by the author and imaged by the participant. A 60 second baseline measure was taken prior to each script while participants sat quietly with their eyes closed. Between stage pauses of approximately ten seconds occurred after each script stage during which participants were instructed to open their eyes. Participants were asked to keep their eyes closed during imagery presentation and to concentrate on imagery details currently being described. Each stage was approximately sixty seconds in duration. VASs were completed at the conclusion of each script, with the key elements of each stage read by the researcher to ensure stage specific ratings. In order to overcome order effects, script presentation was counterbalanced between subjects. Participants were not informed about the order in which the scripts were to be presented in order to reduce anticipatory responses. Each step of the procedure was carefully explained before it occurred and experimental debriefing was provided at the end of each session.

7.2.4 Design and data analysis

The study employed a 3 x 4 x 4 mixed factorial design with repeated measures. The between subjects factor was group (PTSD, ASD, subclinical); and the within subjects factors were script type (LAN, HAN, MVA, Post MVA) and script stage (setting the scene, approach, incident, consequence). The dependent variables were the psychophysiological and psychological responses to imagery. Responses to imagery were analyzed using repeated measures ANOVA with Huynh-Feldt correction, and with Fisher LSD post hoc analyses. A significance level of .05 was adopted for all analyses. MANOVA could not be used due to the ratio of participants to dependent variables. Imagery ability data were analyzed for

group differences using one way ANOVA, and PDI data were analyzed using chi square analysis.

7.2.5 Transformation and scoring of psychophysiological data

Data were obtained from a 30 second period of each stage of each script and baseline, which was consistently selected from the 15 second to 45 second time mark of each script stage. This specific time frame was implemented to ensure accurate between subject comparisons. RESP was measured in breaths per minute, mean SCL and HR (beats per minute) were calculated, and EMG was calculated from an integrated measure. The FBV scores represented changes from baseline, given that the direction of blood volume change is more meaningful than the mean stage response for this measure (e.g., Stern et al., 1980). The validity of these methods of scoring has been demonstrated by various studies (e.g., Brain et al., 1998; Haines et al., 1995).

7.3 Results

7.3.1 Overview

Imagery ability was compared between groups as a control measure. Between group comparisons of psychophysiological and psychological responses to imagery were then presented. Given the considerable amount of data, some results have been included in the appendices, with the most illustrative data included in the main body of this study. The analyses were concluded with a between group examination of peritraumatic fear of death and injury.

7.3.2 Imagery ability

There were no significant between group differences in imagery ability, F (2,48) = 1.4, $p > .05$, as measured by the Betts QMI Vividness of Imagery Scale (Sheehan, 1967). The mean imagery ability score of the sample ($N = 51$) was 77.60 with a standard deviation of 22.03, reflecting the adequate ability of participants to image clearly and vividly.

7.3.3 Psychophysiological responses to imagery

Script by stage by group interaction - FBV

A significant script by stage by group interaction was demonstrated for FBV, F (2,48) = 2.59, $p < .05$. The FBV means and standard deviations for each stage of each script are presented in Appendix D-3. Decreased FBV from baseline, identified by a positive FBV change score, demonstrates a reduction in FBV that is indicative of increased psychophysiological tension. Conversely, increased FBV from baseline, identified by a negative FBV change score, demonstrates increased FBV that is indicative of decreased psychophysiological tension. Therefore, when graphically presented, a higher score is indicative of increased arousal.

Between group differences. Differences in FBV change scores between groups at each stage of each script were examined. Due to the considerable amount of data, in the interests of brevity, post hoc statistics are presented in full in Appendix D-4. At stage four of the MVA script, the PTSD group had significantly decreased FBV from baseline in comparison to the subclinical (*Fisher LSD* = 5.08, $p < .05$) and the ASD groups (*Fisher LSD* = 5.24, $p < .05$). Figure 3 displays the between groups across stages FBV mean change score responses to the MVA script. At stage two

of the Post MVA script, the ASD group had significantly decreased FBV from baseline in comparison with the subclinical (*Fisher LSD* = 2.89, $p < .05$) and the PTSD groups (*Fisher LSD* = 2.82, $p < .05$). Figure 4 displays the between groups across stages FBV mean change score responses to the Post MVA script. There were no significant between group differences in FBV change scores for the other stages of the MVA and Post MVA scripts, or the stages of the HAN and LAN scripts.

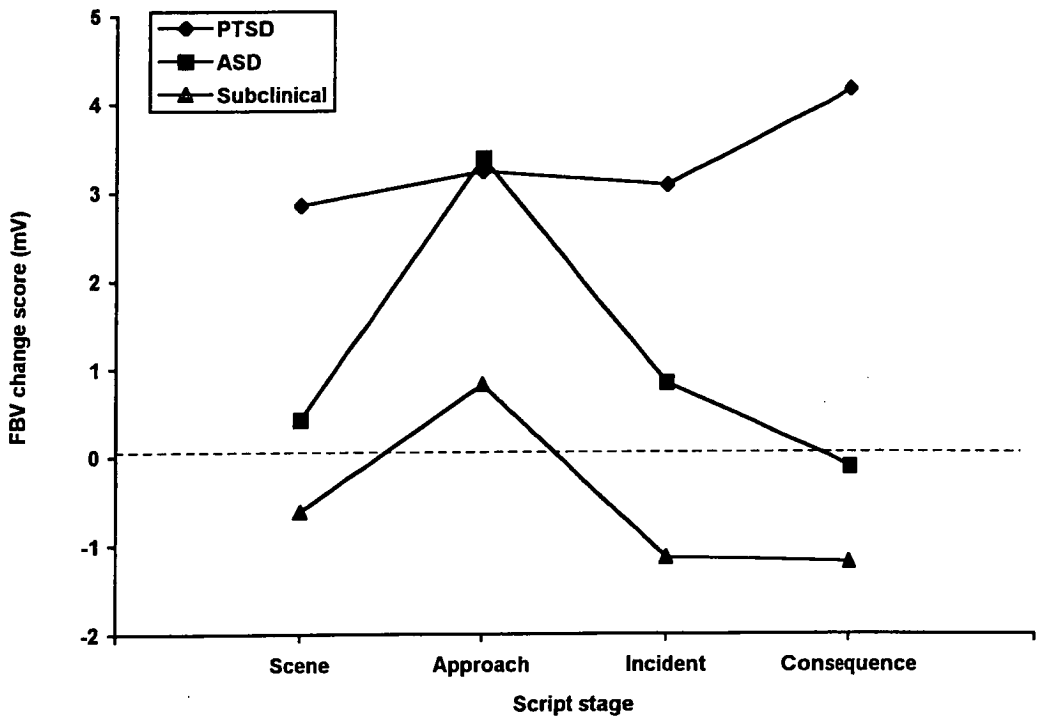

Figure 3.

FBV (mV) mean change scores between groups across stages in response to the MVA script ($N = 51$).

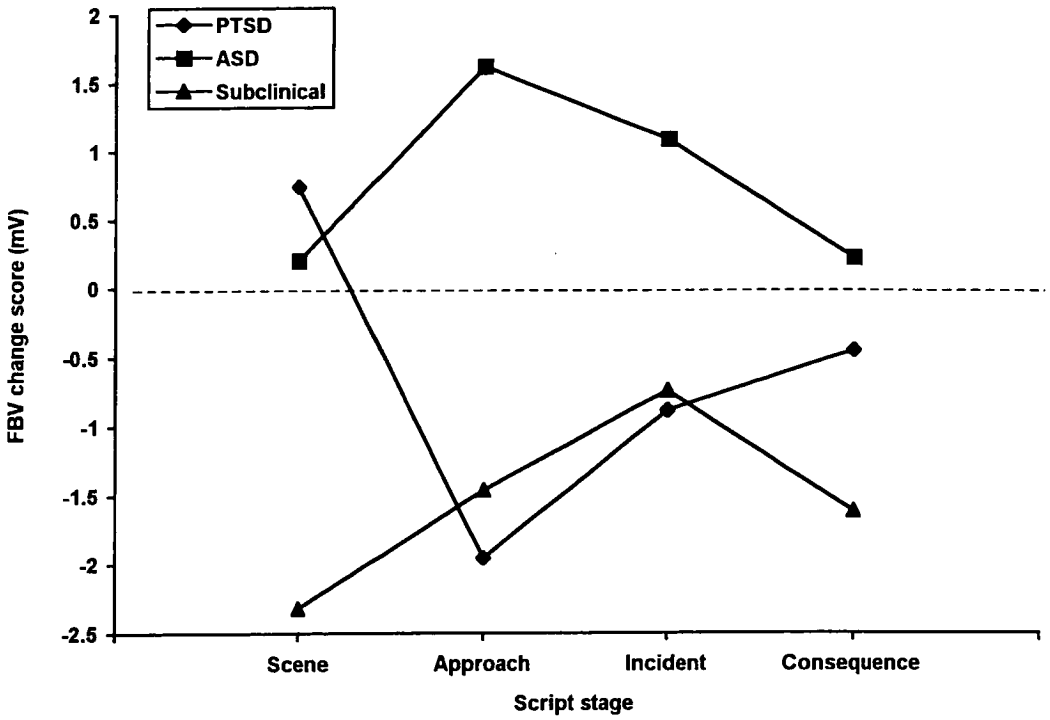

Figure 4.

FBV (mV) mean change scores between groups across stages in response to the Post MVA script ($N = 51$).

Between script differences for each group. Analyses of between script differences at each stage for each group were calculated separately. The post hoc statistics are presented in full in Appendix D-5. Post hoc analyses demonstrated that for the PTSD group, the mean FBV change scores calculated in response to stage one (*Fisher LSD* = 1.39, $p < .05$) and stage three (*Fisher LSD* = 2.64, $p < .05$) of the MVA script were significantly greater than the corresponding LAN, HAN and Post MVA stage measures. In addition, the mean FBV change score of the PTSD group calculated in response to stage four of the MVA script was significantly greater than the Post MVA stage four score (*Fisher LSD* = 2.86, $p < .05$). There were no

significant differences in FBV change scores from baseline between scripts at each stage for the ASD and subclinical groups.

Across stage differences for each group. Differences across the stages of each script for each group were calculated. Post hoc statistics are presented in full in Appendix D-6. Fisher LSD analyses demonstrated that the mean FBV change score calculated in response to stage two of the MVA script was significantly greater for the ASD group than the other stages of this script, reflecting significantly reduced FBV indicative of greater psychophysiological tension for the ASD group at this script stage (*Fisher LSD* = 1.91, $p < .05$). Fisher LSD analyses also demonstrated that for the subclinical group, the mean FBV change score for stage two was significantly greater than those of the other MVA stages for this group (*Fisher LSD* = 1.91, $p < .05$). There were no significant differences in mean FBV change scores across stages of any script for the PTSD group; or for the LAN, HAN, and Post MVA scripts for the ASD and subclinical groups.

Script by stage interaction - HR

A significant script by stage interaction was demonstrated for HR, F (2,48) = 3.05, $p < .005$. The HR means for each stage of each script are presented in Figure 5. The standard deviations for each stage of each script for the total sample are presented in Appendix D-7. Means and standard deviations for each group across the stages of each script for HR are presented in Appendix D-8. No significant baseline differences in HR were found between groups or scripts. Baseline data and analyses are presented in Appendix D-9.

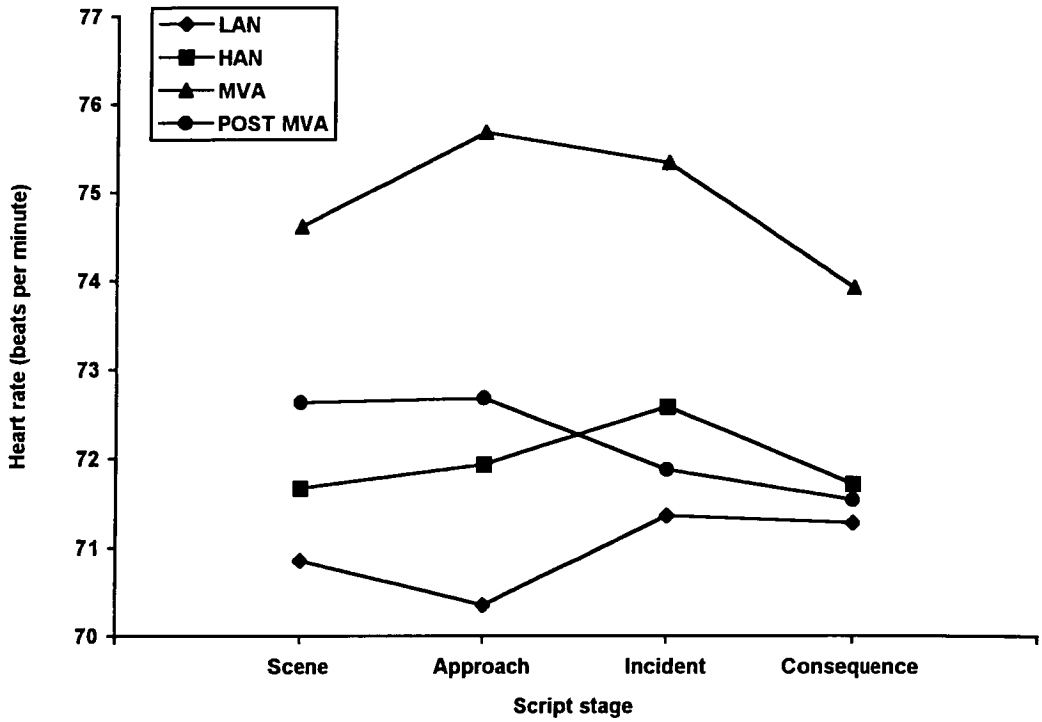

Figure 5.

Mean HR (bpm) across the stages of each script for the total sample (N=51).

Between script differences at each stage. Differences in mean HR between scripts at each stage were examined. Post hoc statistics are presented in full in Appendix D-10. Significant between script differences in mean HR at each stage were demonstrated. Mean HR in response to all four stages of the MVA script were significantly higher than the mean HR responses to the four stages of the other three scripts (stage one *Fisher LSD* = 1.41, $p < .05$; stage two *Fisher LSD* = 1.4, $p < .05$; stage three *Fisher LSD* = 1.44, $p < .05$; stage four *Fisher LSD* = 1.27, $p < .05$). The other significant differences were higher mean HR in response to the Post MVA script in comparison to the LAN script at stage one (*Fisher LSD* = 1.4,

p <.05) and stage two (*Fisher LSD* = 1.4, p < .05); and higher mean HR in response to stage two of the HAN script in comparison to the LAN script (*Fisher LSD* = 1.3, p < .05).

Across stage differences for each script. Differences in mean HR across the stages of each script were examined. Post hoc statistics are presented in full in Appendix D-11. Mean HR during the MVA script was significantly higher during stages 2 and 3 than during stages 1 and 4 (*Fisher LSD* = .95, p < .05). There were no significant across stage differences in mean HR for the other three scripts.

Other psychophysiological measures

RESP. A significant main effect for RESP was demonstrated for group, $F(2,48)= 4.37, p < .05$. Post hoc analyses demonstrated that the RESP (breaths per minute) of the ASD group (M = 20.86, SD = 4.20) was significantly higher than the PTSD (M = 15.52, SD = 4.25) and subclinical groups (M = 15.75, SD = 3.13). Means and standard deviations for each group across the stages of each script for RESP are presented in Appendix D-12. No significant baseline differences in RESP were found between groups or scripts. Baseline data and analyses are presented in Appendix D-13.

EMG. A significant main effect for EMG was found for group across scripts, F (2,48) = 2.99, p < .05, and at baseline, F (2,48) = 3.20, p = .049. Post hoc analyses demonstrated that mean EMG (mV) of the PTSD group (M = 725.36, SD = 590.35) was significantly higher than the ASD (M = 353.26, SD = 288.65) and subclinical groups (M = 445.35, SD = 290.66) across scripts, and mean EMG of the PTSD was significantly higher than the ASD group at baseline. Means and

standard deviations for each group across the stages of each script for EMG are presented in Appendix D-14. No significant baseline differences in EMG were found between scripts. Baseline data and analyses are presented in Appendix D-15.

SCL. No significant between group or script differences were found for SCL between scripts, groups or at baseline. The means and standard deviations for each group across the stages of each script for SCL are presented in Appendix D-16. Baseline data and analyses are presented in Appendix D-17.

7.3.4 Psychological responses to imagery

Results relating to VASs controlling for script accuracy, imagery clarity and freedom from distractibility were within acceptable limits. No significant between group differences were evident for these measures. Descriptive data and analyses for these scales are presented in Appendix D-18.

Script by stage by group interactions

Significant script by stage by group interactions were demonstrated for the following scales: relaxed/tense, F (2,48) = 3.01, $p < .001$; calm/angry, F (2,48) = 2.10, $p < .05$; not guilty/guilty, F (2,48) = 2.46, $p < .05$; and normal/numb, F (2,48) = 2.35, $p < .05$. In addition, a significant interaction which became a trend following Huynh-Feldt correction was found for the normal/unreal scale, F (2,48) = 1.79, $p = .07$. The means and standard deviations for each stage of each script for these measures are presented in Appendix D-19.

Between group differences. Differences in mean VAS scores between groups at each stage of each script were examined. Post hoc statistics are presented in Appendix D-20.

Relaxed/Tense: At stage three of the HAN script the ASD and PTSD groups rated significantly more tension than the subclinical group (ASD/subclinical *Fisher LSD* = 16.78, $p < .05$, PTSD/subclinical *Fisher LSD* = 15.92, $p < .05$), as displayed in Figure 6. At stages two and four of the Post MVA script the subclinical group rated significantly more tension than the PTSD group (stage two: subclinical/PTSD *Fisher LSD* = 16.54, $p < .05$; stage four: subclinical/PTSD *Fisher LSD* = 15.98, $p < .05$), as displayed in Figure 7. There were no significant between group differences in subjective tension at each stage of the LAN and MVA scripts.

Calm/Angry: At stage four of the Post MVA script the subclinical group rated significantly more anger than the PTSD group (*Fisher LSD* = 6.47, $p < .05$). These results are displayed in Figure 8. There were no other significant between group differences in subjective anger at any other stage across the four scripts.

Not guilty/ Guilty: At stage four of the MVA script the ASD group rated significantly greater feelings of guilt than the subclinical (*Fisher LSD* = 20.84, $p < .05$) and PTSD groups (*Fisher LSD* = 19.96, $p < .05$). These results are displayed in Figure 9. There were no other significant between group differences in subjective guilt at any other stage across the four scripts.

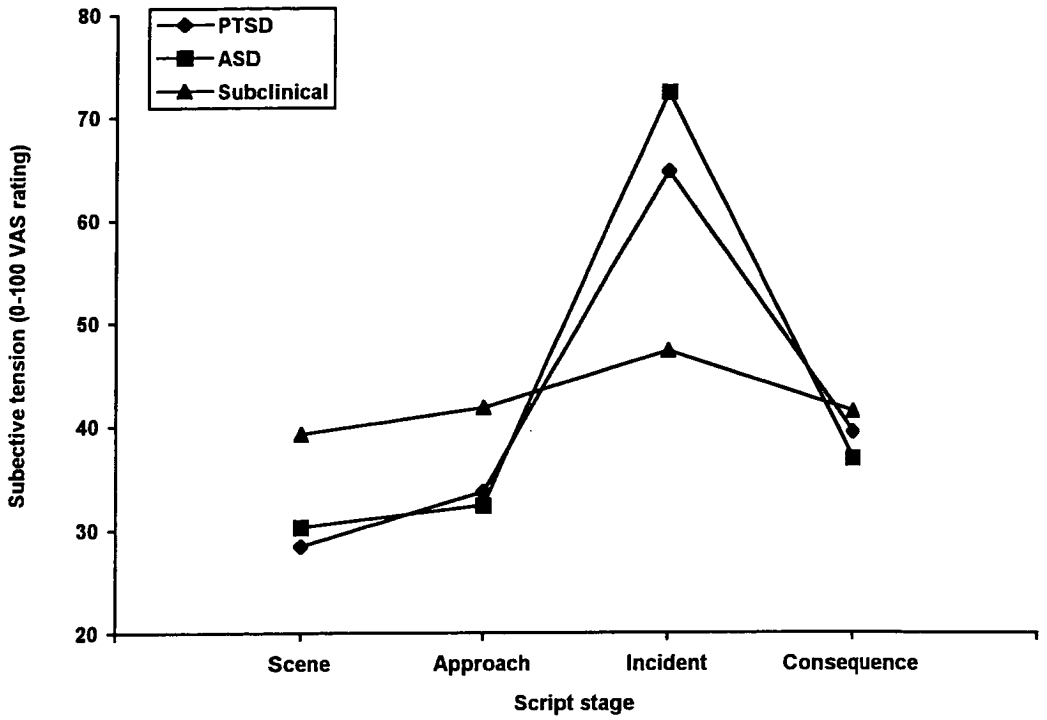

Figure 6.

Mean VAS ratings of subjective tension between groups across stages in response to the HAN script ($N = 51$).

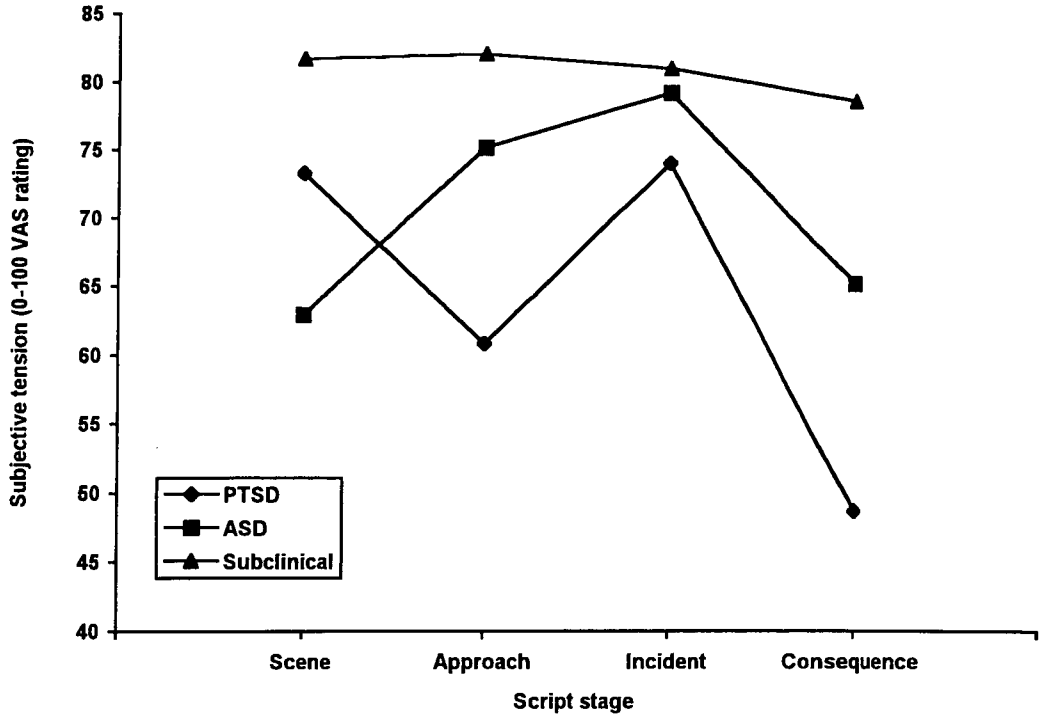

Figure 7.

Mean VAS ratings of subjective tension between groups across stages in response to the Post MVA script ($N = 51$).

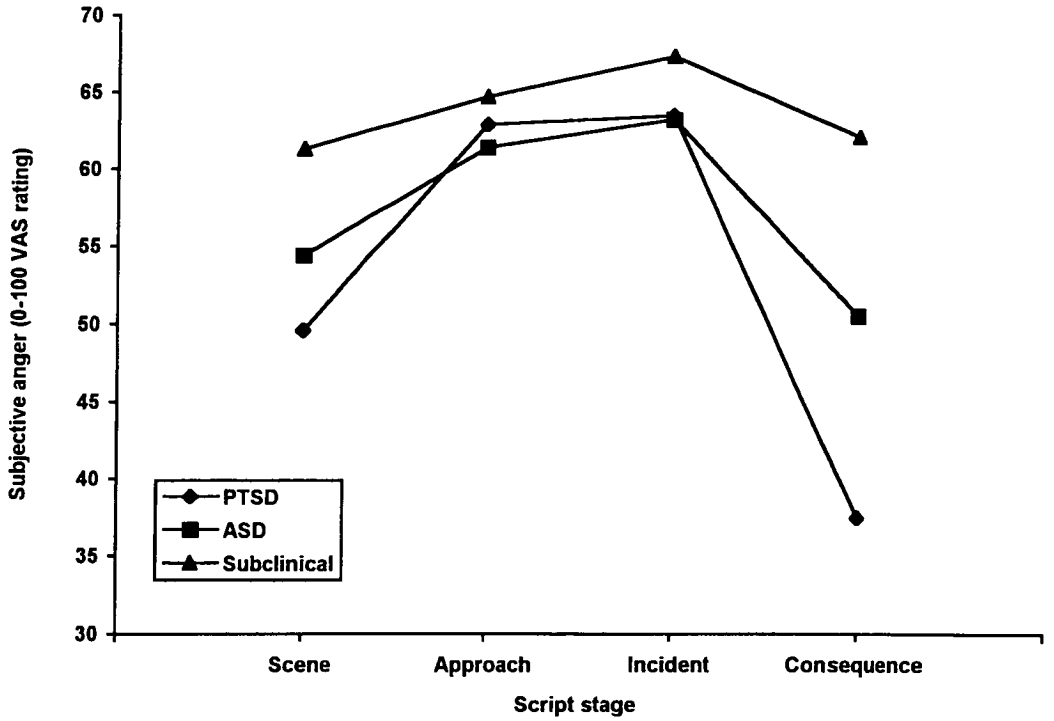

Figure 8.

Mean VAS ratings of subjective anger between groups across stages in response to the Post MVA script ($N = 51$).

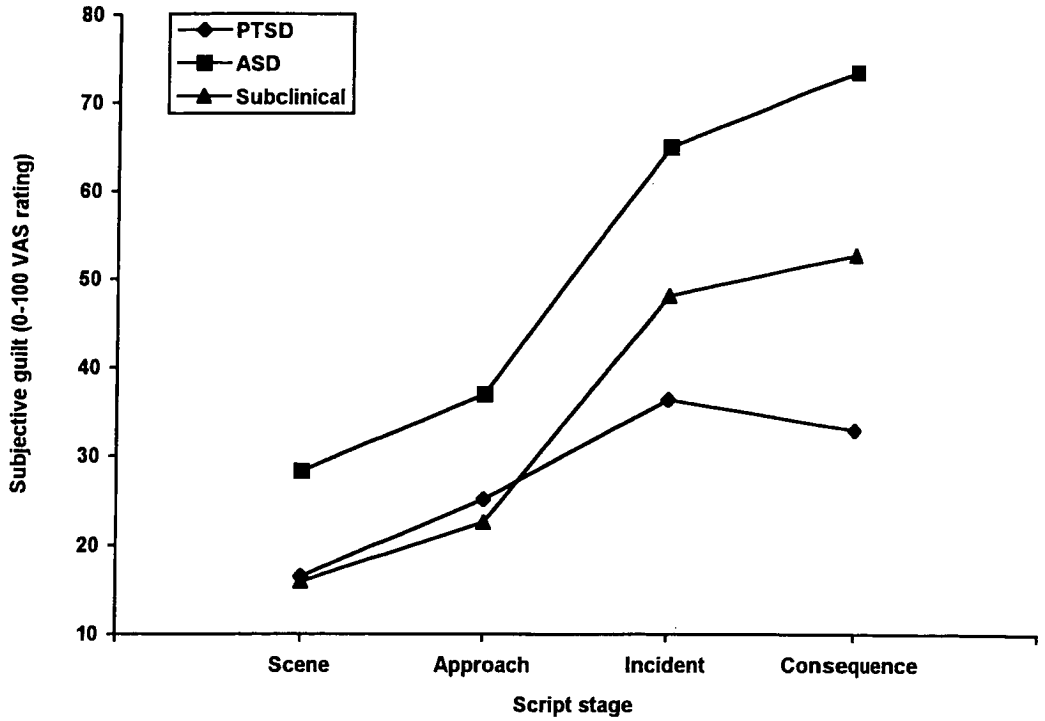

Figure 9.

Mean VAS ratings of subjective guilt between groups across stages in response to the MVA script ($N = 51$).

Normal/Numb: At stages three and four of the HAN script the PTSD and ASD groups rated significantly greater feelings of numbness than the subclinical group (stage three: PTSD/subclinical *Fisher LSD* = 12.65, $p < .05$, ASD/subclinical *Fisher LSD* = 13.34, $p < .05$; stage four: PTSD/subclinical *Fisher LSD* = 11.40, $p < .05$, ASD/subclinical *Fisher LSD* = 12.02, $p < .05$). These results are presented in Figure 10. At stages three and four of the MVA script the ASD group rated significantly greater feelings of numbness than the PTSD and subclinical groups (stage three: ASD/PTSD *Fisher LSD* = 18.47, $p < .05$, ASD/subclinical *Fisher LSD* = 19.28, $p < .05$; stage four: ASD/PTSD *Fisher LSD* = 18.89, $p < .05$, ASD/subclinical *Fisher LSD* = 19.72, $p < .05$). These results are

presented in Figure 11. There were no other significant between group differences in subjective numbness at any other stage across the four scripts.

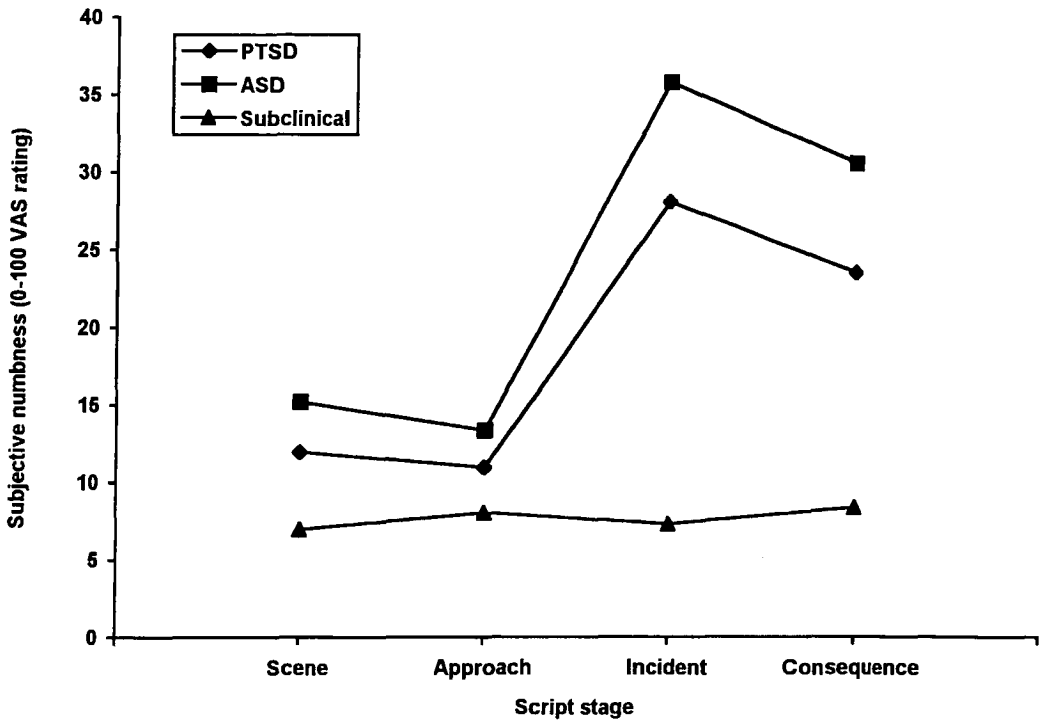

Figure 10.

Mean VAS ratings of subjective numbness between groups across stages in response to the HAN script ($N = 51$).

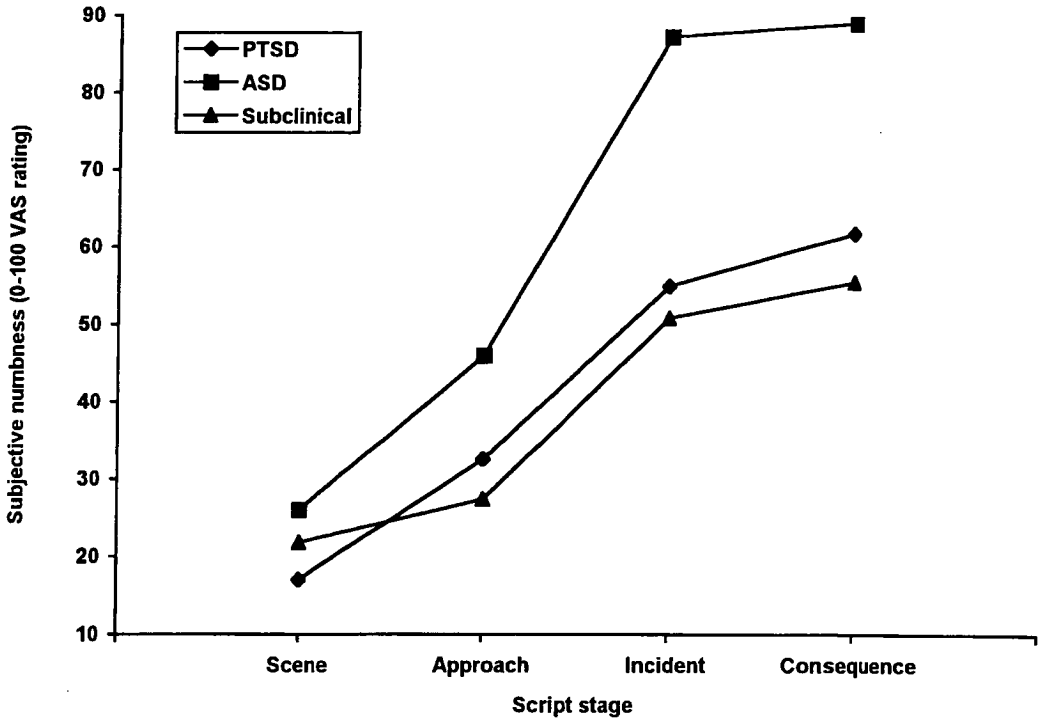

Figure 11.

Mean VAS ratings of subjective numbness between groups across stages in response to the MVA script ($N = 51$).

Normal/Unreal: At stages three and four of the HAN script the ASD group rated significantly greater feelings of unreality than the subclinical group (stage three: ASD/subclinical *Fisher LSD* = 10.17, $p < .05$; stage four: ASD/subclinical *Fisher LSD* = 17.31, $p < .05$). These results are displayed in Figure 12. At stage four of the MVA script the ASD group rated significantly greater feelings of unreality than the subclinical group (ASD/subclinical *Fisher LSD* = 19.78, $p < .05$). At stage three of the MVA script there was a trend towards significance ($p = .054$) for the ASD group to rate greater feelings of unreality than the PTSD and subclinical groups (ASD/PTSD *Fisher LSD* = 17.94, $p < .05$, ASD/subclinical *Fisher LSD* = 18.73, $p < .05$). These results are displayed in Figure 13. There were

no other significant between group differences in feelings of subjective unreality at any other stage across the four scripts.

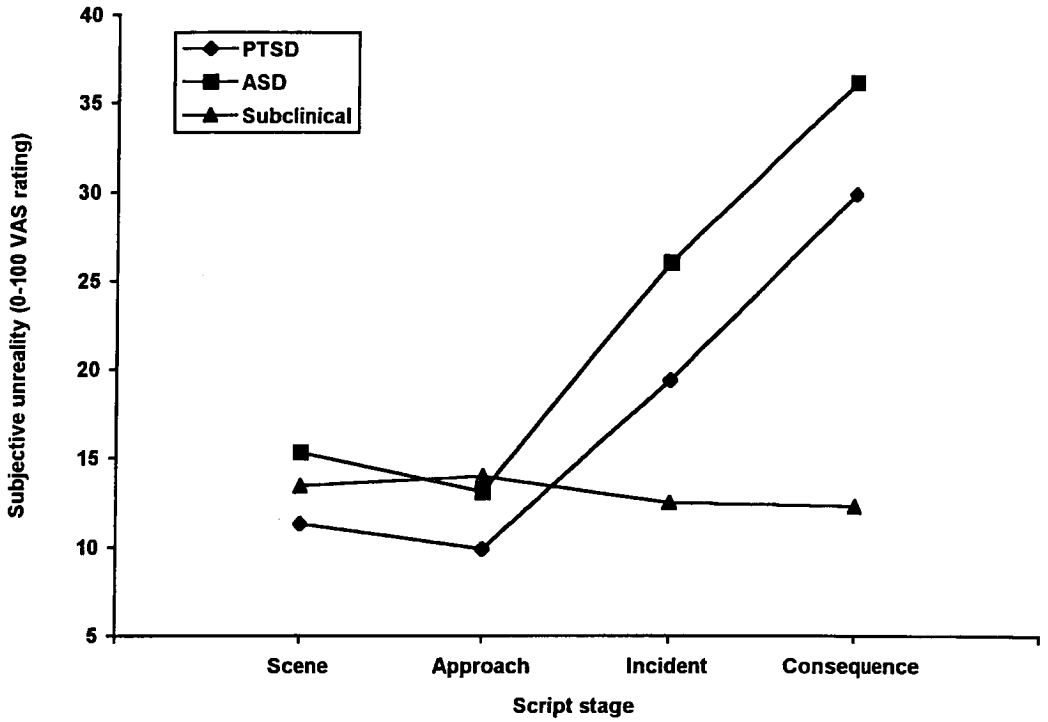

Figure 12.

Mean VAS ratings of subjective unreality between groups across stages in response to the HAN script ($N = 51$).

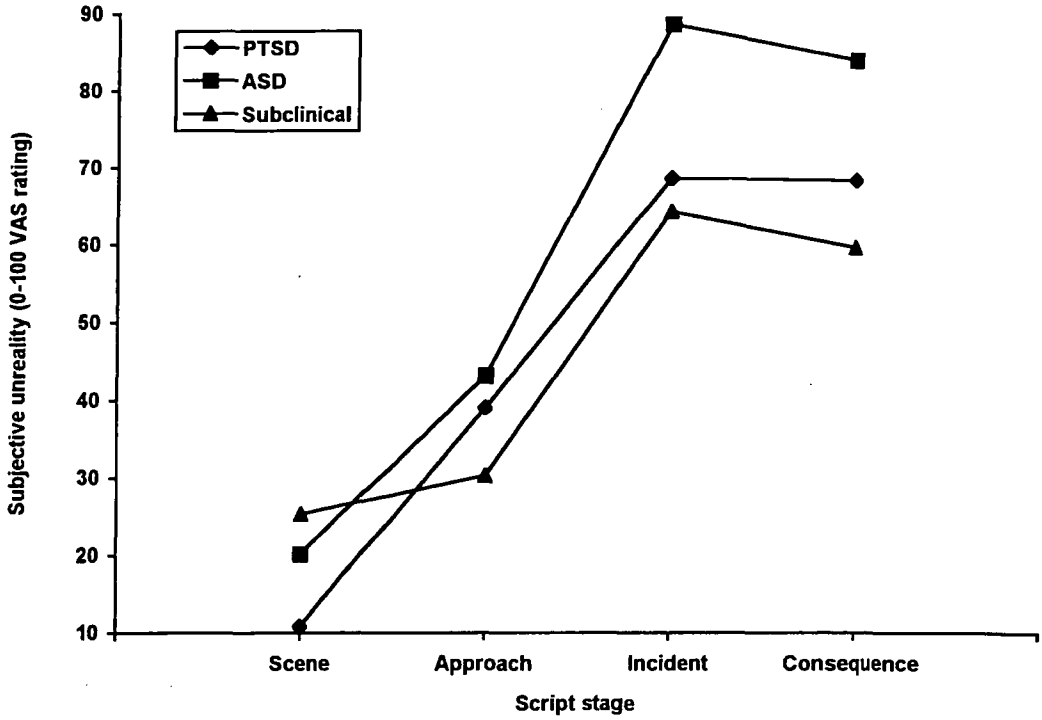

Figure 13.

Mean VAS ratings of subjective unreality between groups across stages in response to the MVA script ($N = 51$).

Between script differences for each group. Analyses of between script differences at each stage for each group were calculated separately. Table 13 presents the post hoc statistics. Due to the large number of significant comparisons, directions of significant differences are presented in the table using the first letter of each script type. The majority of the comparisons reflect the pattern Post MVA, MVA, HAN and LAN in descending VAS ratings, with three exceptions for which the MVA rating was higher than the other scripts. The exceptions applied to ratings of subjective tension by the PTSD group at the consequence stage, and the ratings of subjective numbness by the ASD group at the incident and consequence stages.

Table 13.

Post hoc statistics for between script differences in VAS ratings at each stage for each group (df = 2, 48).

Group	Stage	*F*	*p*	*LSD*	*Script differences*
Dimension					
Relaxed/Tense					
PTSD	Scene	25.47	<.0005	15.54	P>M,H>L
	Approach	15.45	<.0005	17.28	P,M>H>L
	Incident	38.53	<.0005	14.69	P,M,H>L
	Consequence	26.14	<.0005	15.74	M>P,H>L
ASD	Scene	12.35	<.0005	17.83	P>M,H>L
	Approach	9.05	<.0005	22.02	P,M>H,L
	Incident	32.04	<.0005	32.04	P,M,H>L
	Consequence	18.11	<.0005	16.82	P,M>H,L
Subclinical	Scene	20.64	<.0005	17.39	P>M,H>L
	Approach	27.55	<.0005	16.36	P>M,H>L
	Incident	58.06	<.0005	12.98	P,M>H>L
	Consequence	36.86	<.0005	15.47	P,M>H>L

(Table continues...)

Table 13 (continued...)

Group	Stage	*F*	*p*	*LSD*	*Script differences*
Dimension					
Calm/Angry					
PTSD	Scene	10.74	<.0005	14.98	P>M,H,L
	Approach	20.81	<.0005	14.39	P>M,H>L
	Incident	18.31	<.0005	15.63	P>M,H>L
	Consequence	11.64	<.0005	15.37	P,M>H>L
ASD	Scene	14.24	<.0005	14.76	P>M,H>L
	Approach	14.76	<.0005	16.05	P>M>H,L
	Incident	19.25	<.0005	16.62	P,M>H>L
	Consequence	32.69	<.0005	12.46	P,M>H,L
Subclinical	Scene	23.76	<.0005	12.96	P>M>H,L
	Approach	16.87	<.0005	15.58	P>M>H,L
	Incident	39.99	<.0005	12.29	P,M>H,L
	Consequence	19.43	<.0005	16.59	P,M>H>L
Not guilty/Guilty					
PTSD	Scene	24.05	<.0005	11.99	P>M,H,L
	Approach	25.05	<.0005	13.88	P>M,H,L
	Incident	23.91	<.0005	14.26	P>M>H,L
	Consequence	26.61	<.0005	14.76	P>M>H,L

(Table continues...)

Table 13 (continued...)

Group	Stage	*F*	*p*	*LSD*	*Script differences*
Dimension					
ASD	Scene	18.60	<.0005	15.76	P>M,H>L
	Approach	24.66	<.0005	16.46	P>M>H,L
	Incident	46.34	<.0005	14.55	P,M>H,L
	Consequence	49.80	<.0005	14.33	P,M>H,L
Subclinical	Scene	21.06	<.0005	14.78	P>M,H,L
	Approach	16.29	<.0005	16.41	P>M,H,L
	Incident	18.98	<.0005	17.15	P,M>H,L
	Consequence	17.19	<.0005	18.35	P,M>H,L
Normal/Numb					
PTSD	Scene	56.68	<.0005	10.99	P>M,H,L
	Approach	37.14	<.0005	13.67	P>M>H,L
	Incident	15.78	<.0005	17.84	P,M>H>L
	Consequence	27.51	<.0005	16.13	P,M>H>L
ASD	Scene	28.50	<.0005	13.33	P>M,H>L
	Approach	22.74	<.0005	15.60	P>M>H,L
	Incident	58.04	<.0005	13.26	M>P>H>L
	Consequence	46.97	<.0005	15.33	M>P>H>L

(Table continues...)

Table 13 (continued...)

Group	Stage	*F*	*p*	*LSD*	*Script differences*
Dimension					
Subclinical	Scene	35.92	<.0005	11.67	P>M>H,L
	Approach	32.45	<.0005	12.17	P>M>H,L
	Incident	36.41	<.0005	13.49	P,M>H,L
	Consequence	27.40	<.0005	13.68	P,M>H,L
Normal/Unreal					
PTSD	Scene	72.51	<.0005	10.77	P>M,H,L
	Approach	15.66	<.0005	18.68	P>M>H,L
	Incident	51.87	<.0005	13.85	P,M>H,L
	Consequence	28.76	<.0005	16.71	P,M>H>L
ASD	Scene	19.05	<.0005	14.43	P>M,H,L
	Approach	11.34	<.0005	19.55	P>M>H,L
	Incident	32.60	<.0005	16.38	P>M>H,L
	Consequence	20.85	<.0005	18.79	P,M>H,L
Subclinical	Scene	41.03	<.0005	13.12	P>M,H,L
	Approach	26.82	<.0005	13.64	P>M>H,L
	Incident	22.51	<.0005	16.82	P,M>H,L
	Consequence	23.75	<.0005	16.14	P,M>H,L

Across stage differences for each group. Differences across the stages of each script for each group were calculated. Post hoc statistics are presented in Table 14. Due to the large number of significant comparisons, directions of significant differences are presented in the table using stage numbers.

The comparisons demonstrated significant differences in VAS ratings of tension, anger, guilt, numbness, and unreality across the stages of each script for each group, representing numerous fluctuations in psychological response during the course of each recalled event.

Table 14.

Post hoc statistics for across stage differences in VAS ratings in response to each script by each group (df = 2, 48).

Dimension					
Group	Stage	*F*	*p*	*LSD*	*Stage differences*
Relaxed/Tense					
PTSD	LAN	1.73	n.s.	n.a.	
	HAN	14.58	<.0005	11.44	S3>1,2,4
	MVA	44.73	<.0005	10.35	S3,4>2>1
	Post MVA	7.88	<.0005	12.11	S1,3>4>2
ASD	LAN	2.85	n.s.	n.a.	
	HAN	17.89	<.0005	13.90	S3>1,2,4
	MVA	19.48	<.0005	15.08	S3,4>2>1
	Post MVA	6.03	<.005	9.06	S2,3>1,4
Subclinical	LAN	2.86	n.s.	n.a.	
	HAN	2.69	n.s.	n.a.	
	MVA	28.87	<.0005	11.17	S3,4>2>1
	Post MVA	0.66	n.s.	n.a.	

(Table continues...)

Table 14 (continued...)

Group	Stage	*F*	*p*	*LSD*	*Stage differences*
Dimension					
Calm/Angry					
PTSD	LAN	1.12	n.s.	n.a.	
	HAN	8.59	<.0005	7.99	S3>1,2,4
	MVA	21.48	<.0005	9.90	S3,4>1,2
	Post MVA	7.93	<.0005	12.48	S2,3>1,4
ASD	LAN	4.15	<.05	6.56	S2>1,3,4
	HAN	6.68	<.001	8.47	S3>1,2,4
	MVA	16.88	<.0005	12.09	S3,4>1,2
	Post MVA	4.34	<.01	8.16	S2,3>1,4
Subclinical	LAN	0.80	n.s.	n.a.	
	HAN	1.73	n.s.	n.a.	
	MVA	11.92	<.0005	10.05	S3,4>1,2
	Post MVA	1.68	n.s.	n.a.	
Not guilty/Guilty					
PTSD	LAN	2.28	n.s.	n.a.	
	HAN	3.19	<.05	4.21	S1,2>3,4
	MVA	6.76	<.001	11.03	S3,4>1,2
	Post MVA	8.28	<.0005	4.67	S3>2,4>1

(Table continues...)

Table 14 (continued...)

Group	Stage	*F*	*p*	*LSD*	*Stage differences*
Dimension					
ASD	LAN	0.97	n.s.	n.a.	
	HAN	4.25	<.05	5.84	S1,2>3,4
	MVA	14.11	<.0005	17.26	S3,4>1,2
	Post MVA	2.36	n.s.	n.a.	
Subclinical	LAN	1.26	n.s.	n.a.	
	HAN	1.72	n.s.	n.a.	
	MVA	18.06	<.0005	12.19	S3,4>1,2
	Post MVA	0.93	n.s.	n.a.	
Normal/Numb					
PTSD	LAN	1.53	n.s.	n.a.	
	HAN	8.67	<.0005	7.88	S3,4>1,2
	MVA	28.07	<.0005	12.44	S3,4>2>1
	Post MVA	2.22	n.s.	n.a.	
ASD	LAN	7.87	<.0005	1.52	S1>2>3>4
	HAN	9.75	<.0005	10.08	S3,4>1,2
	MVA	41.98	<.0005	13.84	S3,4>2>1
	Post MVA	2.07	n.s.	n.a.	

(Table continues...)

Table 14 (continued...)

Group	Stage	*F*	*p*	*LSD*	*Stage differences*
Dimension					
Subclinical	LAN	2.48	n.s.	n.a.	
	HAN	1.42	n.s.	n.a.	
	MVA	22.90	<.0005	10.77	S3,4>1,2
	Post MVA	4.39	<.01	8.32	S1,2,3>4
Normal/Unreal					
PTSD	LAN	2.16	n.s.	n.a.	
	HAN	5.95	<.005	9.27	S4>1,2,3
	MVA	32.95	<.0005	13.38	S3,4>2>1
	Post MVA	2.99	<.05	10.39	S1,3,4>2
ASD	LAN	2.14	n.s.	n.a.	
	HAN	8.68	<.0005	10.52	S3,4>1,2
	MVA	39.95	<.0005	15.02	S3,4>2>1
	Post MVA	2.83	<.05	5.20	S4>1,2,3
Subclinical	LAN	3.20	<.05	2.60	S1,2>3,4
	HAN	2.45	n.s.	n.a.	
	MVA	21.41	<.0005	12.88	S1,2>3,4
	Post MVA	6.91	<.001	9.03	S1,2>3,4

Script by stage interactions

Significant script by stage interactions were demonstrated for the following VASs: happy/sad, F (2,48) = 28.04, p < .0005; unafraid/afraid, F (2,48) = 32.23, p

$< .0005$; and comfortable/uncomfortable, $F(2,48) = 34.11$, $p < .0005$. The means and standard deviations for each stage of each script for these measures are presented in Table 15. The means and standard deviations for each group across each stage of each script for these measures are presented in Appendix D-21.

The table shows that VAS ratings of sadness, fear and discomfort were all significantly greater in response to the trauma scripts at all stages than the neutral scripts, and that the Post MVA script most often evoked the greatest ratings on each dimension at each stage.

Table 15.

Means and standard deviations across stages of each script for sadness, fear and discomfort VAS ratings (N = 51).

Dimension								
Script	Scene		Approach		Incident		Consequence	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Happy/Sad								
LAN	16.51	(17.13)	13.72	(14.97)	13.26	(14.44)	13.14	(15.92)
HAN	21.84	(20.21)	20.98	(22.21)	24.46	(22.58)	23.95	(26.97)
MVA	31.55	(23.33)	43.41	(24.24)	66.85	(22.48)	70.32	(21.41)
Post MVA	70.44	(22.38)	69.31	(19.21)	80.30	(16.71)	67.38	(20.45)

(Table continues...)

Table 15 (continued...)

Dimension								
Script	Scene		Approach		Incident		Consequence	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Unafraid/Afraid								
LAN	6.84	(7.62)	6.85	(8.12)	5.82	(6.76)	7.17	(9.36)
HAN	8.83	(8.13)	9.50	(7.84)	8.40	(9.46)	9.90	(9.35)
MVA	25.26	(24.38)	46.10	(30.73)	71.98	(26.15)	47.44	(29.11)
Post MVA	64.20	(26.72)	62.93	(24.24)	65.05	(26.03)	59.59	(27.95)
Comfortable/Uncomfortable								
LAN	7.95	(9.28)	7.31	(8.48)	6.94	(6.98)	5.78	(6.75)
HAN	18.61	(18.89)	24.62	(20.86)	41.37	(28.68)	31.56	(32.00)
MVA	26.79	(24.69)	46.08	(27.37)	75.83	(21.86)	77.08	(21.73)
Post MVA	69.95	(22.84)	76.00	(18.11)	75.45	(18.79)	71.36	(18.62)

Between script differences at each stage. Differences in mean VAS scores between scripts at each stage were examined. Post hoc statistics are presented in Table 16. The direction of significant comparisons is denoted in the table by the first letter of each script type.

Table 16.

Post hoc statistics for between script differences in sadness, fear and discomfort VAS ratings at each stage (df = 2, 48).

Dimension				
Stage	*F*	*p*	*LSD*	*Script differences*
Happy/Sad				
Scene	57.70	<.0005	8.93	P>M>H,L
Approach	62.51	<.0005	8.80	P>M>H,L
Incident	136.56	<.0005	7.78	P>M>H>L
Consequence	79.47	<.0005	9.24	P,M>H>L
Unafraid/Afraid				
Scene	96.85	<.0005	7.51	P>M>H,L
Approach	91.17	<.0005	8.06	P>M>H,L
Incident	157.77	<.0005	7.92	P,M>H,L
Consequence	104.16	<.0005	8.46	P,M>H,L
Comfortable/Uncomfortable				
Scene	98.88	<.0005	7.22	P>M>H,L
Approach	95.33	<.0005	7.97	P>M>H,L
Incident	164.24	<.0005	7.68	P,M>H,L
Consequence	111.20	<.0005	8.23	P,M>H,L

Across stage differences for each script. Differences in mean VAS scores across the stages of each script were examined. Post hoc statistics are presented in Appendix D-22.

Happy/Sad: Mean ratings of sadness were significantly higher in response to stages three and four of the MVA script than stage two, and these were greater than the response to stage one (*Fisher LSD* = 75.52, $p < .0001$).These results are displayed in Figure 14. In response to the Post MVA script, mean ratings of subjective sadness were significantly highest in response to stage three than the other three stages (*Fisher LSD* = 10.15, $p < .0001$). These results are displayed in Figure 15. There were no significant across stage differences in sadness in response to the LAN and HAN scripts.

Unafraid/Afraid: Mean ratings of fear were significantly higher in response to stages three and four of the MVA script than stages one and two of this script, and fear was higher in response to stage two than stage one (*Fisher LSD* = 53.88, $p < .0001$). These results are displayed in Figure 14. There were no significant across stage differences in fear in response to the LAN, HAN and Post MVA scripts.

Comfortable/Uncomfortable: Mean ratings of feeling uncomfortable were significantly higher in response to stages three and four of the MVA script than stage two, and these were greater than the response to stage one (*Fisher LSD* = 53.88, $p < .0001$). These results are displayed in Figure 14. In response to the Post MVA script, mean ratings of feeling uncomfortable were significantly higher in response to stages two, three and four than stage one (*Fisher LSD* = 12.05, $p < .005$). These results are displayed in Figure 15. There were no significant across stage differences in feeling uncomfortable in response to the LAN and HAN scripts.

Figures 14 and 15 display the patterns of psychological response in response to the MVA and Post MVA scripts respectively for these three psychological measures.

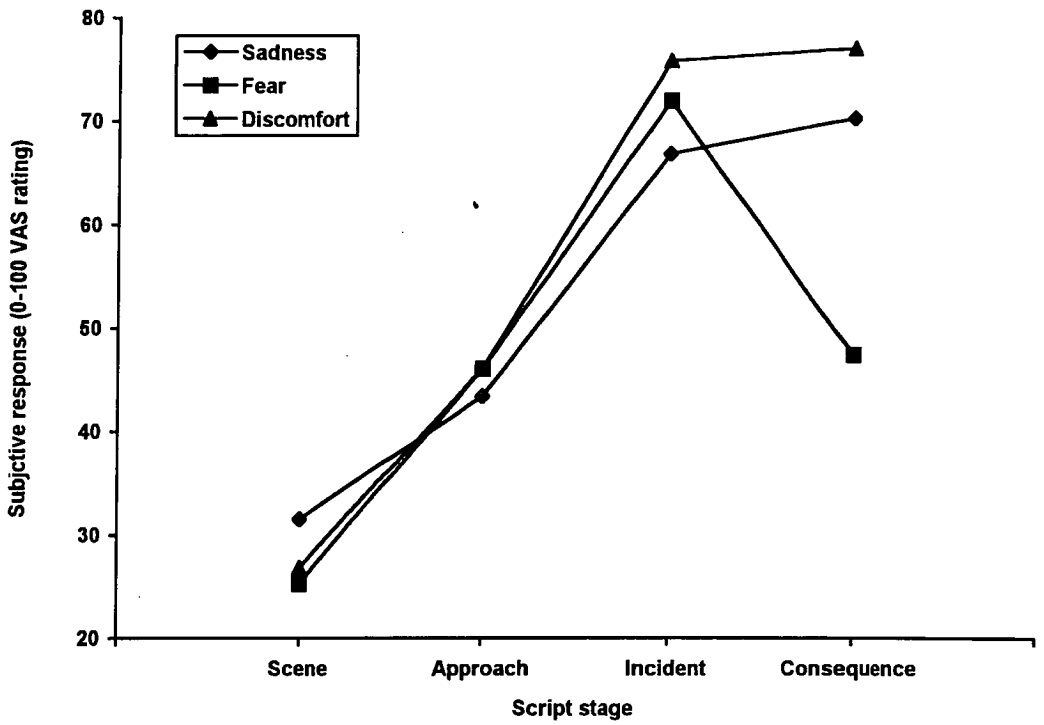

Figure 14.

Mean VAS ratings of subjective sadness, fear and discomfort across stages in response to the MVA script ($N = 51$).

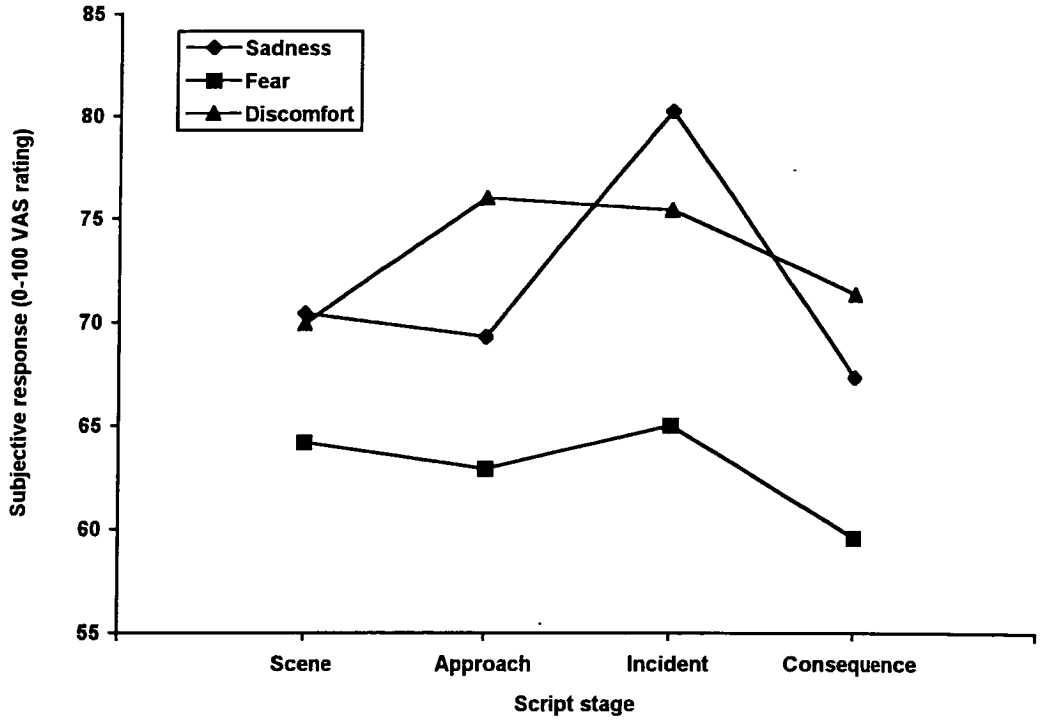

Figure 15.

Mean VAS ratings of subjective sadness, fear and discomfort across stages in response to the Post MVA script (N=51).

In terms of peritraumatic fear of death, there were no significant between group differences in the reported prevalence of experiencing a PDI, χ^2 (2, N = 51) = 4.65, p > .05. The PDI was reported by 27% of the PTSD group, 30% of the ASD group and 28% of the subclinical group. Similarly, there were no between group differences in fear of death of other people involved in the MVA, χ^2 (2, N = 51) = 4.65, p > .05 (PTSD 27%, ASD 30%, subclinical 28%), fear of serious injury to self, χ^2 (2, N = 51) = 5.07, p > .05 (PTSD 53%, ASD 58%, subclinical 52%), or fear of serious injury to others, χ^2 (2, N = 51) = 3.78, p > .05 (PTSD 43%, ASD 46%, subclinical 42%).

7.4 Discussion

7.4.1 Imagery ability

The hypothesis regarding imagery ability was supported, in that there were no between group differences found in imagery ability. This finding, and the nature of the imagery ability scores, indicated that there were no group differences in terms of ability to perform the guided imagery tasks, and that all groups were able to image clearly and effectively as required. Even though this variable was considered to be a control measure, it was deemed necessary to establish support for this hypothesis prior to the interpretation of the data.

7.4.2 Psychophysiological responses to imagery

Personalized traumatic imagery provoked greater psychophysiological reactivity than personalized nontraumatic imagery, as proposed by the second hypothesis. This result is consistent with previous examinations of responses to trauma-related cues (e.g., Blanchard et al., 1986; Keane et al., 1998; Liberzon, Abelson, Flagel, Raz, & Young, 1999; Litz et al., 2000; Malloy et al., 1983; McCaffrey et al., 1993).

The comparison of responses to the MVA versus Post MVA scripts indicated that the MVA script evoked a significantly greater stress response in the psychophysiological modes of FBV and HR, than the Post MVA script for all groups. Between group differences in patterns of arousal across the stages of the trauma scripts were shown by mean FBV responses, also supporting the second hypothesis. Although not statistically significant, it is worth observing that there was a trend for an arousal peak of the PTSD group in this psychophysiological

mode occurring at stage four of the MVA script, when the immediate consequences of the accident were recalled. In comparison, the statistically significant arousal peaks of the ASD and subclinical groups occurred at stage two of the MVA script, when the moments immediately prior to the accident were recalled. The arousal peak of the ASD group was significantly greater than that of the subclinical group in the FBV mode. It may be speculated that the arousal peak of the ASD group at stage two of the MVA script demonstrated awareness of the imminent accident, and consequently reduced the shock of the impact, that may be perceived as a more sudden, shocking experience for the PTSD group.

This difference may have theoretical value in the context of dissociation, in that the higher level of anticipatory arousal of the ASD group may have triggered the onset of a dissociative process. These findings support the third hypothesis. Dissociative processes such as depersonalization are caused by intense distress, and the function of such a process may be distress management and psychological self-protection (e.g., Rothbaum et al., 2001). In terms of script content, dissociative phenomena were predominantly reported during stages three and four of the MVA script, and stage one of the Post MVA script, when the accident and immediate consequences were recalled. It is proposed that the arousal peak of the ASD group at stage two, followed by the arousal reduction throughout MVA script stages three and four and Post MVA stage one provide psychophysiological evidence across time of subjectively reported dissociative experiences. If this proposal is accepted, then this is the first time that the integration of psychophysiological and psychological dissociative processes have been measured throughout the course of recalled MVAs.

Situation specific psychophysiological reactivity was apparent in the HR measure for all groups. Although there were no significant between group differences in heart rate response, this measure supported the use of situation specific scripts to compare psychophysiological reactivity to different events. All groups demonstrated the highest heart rate response to the MVA script. The pattern of arousal across stages, indicated by increased HR, provided further evidence that stages two and three of the MVA script, where the moments just prior to the MVA and the actual impact were recalled, were the most psychophysiologically arousing for all groups. The lack of baseline differences between groups also supported situation specific reactivity of all groups, as opposed to basal hyperarousal associated with specific diagnoses.

Although it is acknowledged that some researchers have found baseline differences in HR (e.g., Blanchard, 1990; Buckley & Kaloupek, 2001), it should be noted that the majority of participants in these studies were male combat veterans in excess of ten years posttrauma. The comparative recency of trauma exposure and the predominantly female sample in the present study may, at least in part, explain the differences in findings, as posttraumatic symptom chronicity and sex differences in cardiac function have been noted as clinically significant variables in the assessment of basal cardiovascular activity (Buckley & Kaloupek, 2001). The findings of the present study support other research that has not found baseline differences (e.g., McFall et al., 1990; Orr et al., 1993; Rothbaum et al., 2001). In a recent review of psychophysiological studies of PTSD, Shalev (1999) also noted that baseline HR and baseline sympathetic activity may not be higher in individuals with PTSD, lending current support for the situation specific reactivity proposal (e.g., Prins, Kaloupek & Keane, 1995).

Group specific modes of psychophysiological response were evident in the modes of EMG and respiration, with comparatively high muscle tension not surprisingly associated with PTSD. It may be speculated that the comparatively high respiration rate of the ASD group may mediate dissociative symptoms. Given that baseline differences were not found between groups or scripts for respiration, the elevated respiration rate of the ASD group situation specific. It may be suggested in light of the previously presented biological theories that rapid breathing is a component of the immediate fight or flight reaction to shock, and that it is this response that plays a role in the induction of dissociative experiences.

It is clear that hyperventilation can occur in response to emotional trauma as well as to physiological stress (Conway, Freeman, & Nixon, 1988). This is particularly true of people who have an anxiety sensitivity (Dowden & Allen, 1997). Indeed, the role of hyperventilation in the experience of panic attacks is well established (Papp et al., 1997), at least in a subtype of Panic Disorder (Hegel & Ferguson, 1997). The experience of hyperventilation has been associated with the negative evaluation of both emotionally arousing and neutral stimuli (Dowden & Allen, 1997). The negative affective evaluation of the MVA that would occur in conjunction with the experience of increased respiratory rate along with the physical sensations that are characteristic of hyperventilation (Skevington, Pilaar, Routh, & MacLeod, 1997) such as paraethesiae in the hands and feet (Mogyoros, Kiernan, Burke, & Bostock, 1997), could trigger a dissociative experience in an individual prone to dissociative states. Support for the view comes from the fact that the peak of arousal for the ASD group was evident at stage two, suggesting that the elements of the MVA were apparent earlier for that group and, apparently, triggered a fear response that lead to dissociation.

There were no between group or script differences found for SCL in response to stimuli or at baseline. This finding reinforced the idiosyncratic nature of psychophysiological response as, despite significant between group and across script and stage differences found for the other measures, this mode of response did not reflect such changes. Due to the latency from stimulus reception to the onset of a SCL response, which has been reportedly greater than that of the other measures (Stern et al., 1980), SCL may not reflect stage by stage responses in the same way as the other psychophysiological measures. As noted by Kaloupek and Bremner (1996), psychophysiological measures are not interchangeable indicators of arousal. These findings reinforce the need for multimodal psychophysiological assessment in order to derive comprehensive clinical information, as recommended by other researchers (e.g., Blanchard & Buckley, 1999). The psychophysiological responses to the four scripts highlighted situation specific responding by all groups, and indicated that recall of the MVA itself provoked greater arousal than the aftermath. Group specific response patterns were evident, and supported the proposed utility of a four stage guided imagery methodology in the assessment of differential posttraumatic responses.

7.4.3 Psychological responses to imagery

Whereas the results of the psychophysiological assessment indicated that greater arousal was elicited during the MVA, different patterns of response were evident when psychological responses to the MVA and its aftermath were examined. When considering the script by stage by group interactions, at stage one, all groups experienced greater negative psychological response to the Post MVA script than they did to the MVA script. This would be expected as stage one

of the MVA script was pre-accident whereas the initial stage of the Post MVA script set the scene for dealing with the immediate aftermath of the MVA.

At stage two, the same pattern of response was evident with the Post MVA script eliciting greater negative response than the MVA script. At stage three it was more usual for the MVA script and the Post MVA script ratings to be equivalent. The Post MVA ratings remained high and the MVA ratings increased to a comparable level coinciding with descriptions of the actual MVA. There were some exceptions. The Post MVA script elicited greater feelings of anger and guilt for the PTSD group than the MVA script. For the ASD group, the MVA script provoked greater feelings of numbness and unreality than the Post MVA script.

At stage four, again, the Post MVA script most commonly elicited similar ratings to the MVA script. There were some exceptions. For the PTSD group, the MVA script provoked greater feelings of tension than the Post MVA script whereas the Post MVA script produced greater feelings of guilt than the MVA script. For the ASD group, the MVA script elicited greater feelings of numbness than did the Post MVA script.

When considering the script by stage interactions, at stage one, the results for the total sample for sadness, fear and discomfort coincided with the results for the individual groups for the other VASs with the Post MVA script eliciting greater negative response than the MVA script. At stage two, the same pattern of response was evident. At stage three, the Post MVA script provoked an equivalent level of fear and discomfort, whereas the Post MVA script elicited greater feelings of sadness than did the MVA script. At stage four, the MVA and Post MVA scripts produced equivalent levels of subjectively reported sadness, fear and discomfort.

The integrated examination of psychophysiological and psychological measures provided evidence of the association between psychophysiological stress reduction and subjective tension reduction, numbness and unreality of the ASD group as the accident was recalled, indicative of the measurement of dissociative phenomena occurring over time. These results support the second, third and fourth hypotheses. Psychological states measured by the VASs reflected group specific patterns of response to posttraumatic cues. In response to stages three and four of the MVA script, the ASD group reported significantly greater dissociative feelings of numbness and unreality than the other groups, as previously noted. These results show that a psychophysiological arousal peak immediately precedes psychological responses associated with dissociative experiences of the ASD group, and support the notion that this study has provided evidence of dissociation occurring during the course of an event. As dissociation has been described as a core feature of ASD (APA, 1994), it is proposed that it may have a self-protective, adaptive role in long term posttraumatic recovery for those individuals who do not develop PTSD following an MVA.

In addition, the ASD group reported significantly greater guilt than the other groups at stage four of the MVA script. This finding may have implications for the role of the attribution of blame in posttraumatic recovery. Self-blame has been proposed to have a protective effect in terms of posttraumatic symptom severity (e.g., Ehlers et al., 1998; Hickling, Blanchard, Buckley, et al., 1999; Ho et al., 2000; Wells et al., 2000; Wenninger & Ehlers, 1998), which may be linked to increased feelings of control when an MVA occurs, rather than the loss of control experienced when another person is responsible for the MVA (Wells et al., 2000).

Therefore, the self-blame of the ASD group may have contributed to reduced symptom severity in the longer term.

It may also be speculated that the anticipatory awareness of the impending MVA impact by the ASD group, reflected by the FBV response, may have allowed this group to respond with cognitive construction of the event. In comparison, the shock of the experience for the PTSD group may have prevented peritraumatic cognitive construction, and this difference may have implications for posttraumatic symptom development. The induction of the feelings of numbness and unreality that may have been caused by this anticipatory response may have been beneficial for the ASD group by protecting them from the immediate psychological impact of the initial threat and the immediate aftermath.

In response to the Post MVA script, the PTSD group reported significantly less tension in response to stages 2 and 4, and less anger in response to stage four than the subclinical group. This tension reduction pattern across time in response to the Post MVA script was consistent with the FBV response. The PTSD group experienced psychophysiological tension reduction following the MVA. This pattern of response corresponds with learning theories of the development of avoidant and phobic responses to anxiety provoking situations as described in chapter three (e.g., Bandura, Blanchard, & Ritter, 1969). It may be interpreted from the data that the PTSD group experienced a sense of relief immediately following the MVA, as they came closer to leaving the scene where they had experienced intense negative emotions. This relief, or tension reduction, may be speculated to provide a mechanism by which avoidance of stimuli related to the MVA scene developed, in accordance with the learning theories described in chapter three. This pattern is not evident in the ASD or subclinical groups. These

results support previous findings that peritraumatic emotional reactions may be predictive of the later development of posttraumatic symptoms (e.g., Bernat at al., 1998; Roemer et al., 1998). This finding may also be linked with the fact that, in the previous study, the PTSD group was found to have a propensity to use avoidant coping strategies. Thus, the tension reduction mechanism associated with avoidance may have been well rehearsed prior to the trauma, in response to other stressors.

There were no significant between group differences between script or across stages in ratings of sadness, fear, or discomfort. The groups consistently reported greater negative feelings in response to the trauma scripts than the neutral scripts. The consistent pattern of response between groups across stages and the comparable intensity of these negative subjective responses reflected that all participants, regardless of diagnostic outcome, perceived their MVA to be equally provocative of feelings of sadness, fear and discomfort. This similarity in emotional interpretation of the event was considered important when analyzing between group differences found in psychophysiological arousal to the MVA script. For example, the PTSD group interpreted the accident subjectively in the same way as the other groups in terms of tension experienced, yet the groups showed different psychophysiological arousal patterns. The data suggests that the level of tension was the same, but the psychophysiological manifestation of the tension differed between groups.

Interestingly, group differences were detected in psychological response to the high arousal nontraumatic exercise script. Between group differences were found in the subjective report of feelings of tension, anger, guilt, numbness and unreality in response to the nontraumatic script content. In response to stages three

and four of the HAN script, the PTSD and ASD groups reported significantly greater numbness than the subclinical group, and the ASD group reported significantly greater feelings of unreality than the subclinical group. The PTSD and ASD groups also reported greater subjective tension than the subclinical group at stage three of the HAN script, where engaging in physical exercise was described. These differences may reflect an association between psychophysiological arousal, tension and dissociation, and are supportive of theories of situation specific differences in psychophysiological responses following trauma (e.g., Blanchard & Hickling, 1997). The results were consistent with a recent study in which highly anxious individuals responded with fear to guided imagery of exercise, despite no significant differences being found in psychophysiological response when compared with a less anxious group (Davis, 2001). These results support the theory of misinterpretation of arousal as fearful and anxiety provoking, concurring with a psychophysiological model of panic attacks (Wilhelm & Margraf, 1997). It may be speculated that the maintenance of posttraumatic symptoms is, in part, related to the subjective misinterpretation of psychophysiological arousal. Given that a feature of posttraumatic stress disorders is hyperarousal, a propensity to misinterpret arousal could reduce wellbeing and contribute to the maintenance of disorder.

There were no significant between group differences in the reported prevalence of experiencing a PDI, thus, the fifth hypothesis was not supported. There were also no between group differences found in fear of death of others or serious injury. Less than one third of each group feared someone else involved in the MVA may die, just over half of each group feared they would be seriously injured, and less than half of each group feared someone else involved would be

seriously injured. These results may be considered interesting, in that the literature indicates that the higher the perceived threat to the physical integrity of self or others, the greater the likelihood of the development of more severe posttraumatic symptoms (e.g., Carson et al., 2000). In light of previous research (e.g., Haines, Williams, Holmes, Wells, et al., 2001) it may be speculated that the PDI is a significant catalyst for a posttraumatic response that may be either positive or negative and, thus, further investigation of associations between the PDI and diagnostic outcomes may be warranted. The psychological responses reflect that a four stage guided imagery methodology has utility in identifying specific patterns of response to situation specific scripts, and facilitates integrated understanding of associations between psychophysiological responses and subjective interpretations of real life events.

7.4.4 Summary and conclusions

The immediate psychophysiological reaction to recall of the MVA indicated that the participants were responding to the immediate threat and were not yet processing other information about the MVA. It is theorized that this was followed in the aftermath of the MVA by an increase in the negative psychological reaction as information processing occurred. This study identified psychophysiological and psychological patterns that may be influential in the differential development of PTSD, ASD and subclinical posttraumatic responses. These findings may assist with vulnerability prediction and early intervention. The four stage guided imagery methodology of integrated psychophysiological and psychological assessment has been evaluated as a clinical tool that may advance current assessment methods of

posttraumatic responses, and may be utilized in treatment approaches. These applications will be further discussed in the final chapter.

The advancement that this methodology demonstrated was the ability to break down one experience into stages, so that patterns of response throughout the experience may be compared. The stage by stage approach facilitated a more specific analysis of the components of an experience than existing methods that have focused on global responses to traumatic stimuli. Although this study exclusively examined MVA trauma, it may be speculated that a four stage guided imagery methodology may be utilized to examine other trauma types and populations.

A strength of this study was the demographic similarities between the groups and stringent exclusion criteria, reducing problems associated with factors such as sex and age differences in psychophysiological response (e.g., Arena, Blanchard, Andrasik, & Myers, 1983). Studies two and three have established that the differential development of PTSD, ASD and subclinical responses to MVA trauma is associated with cognitive, affective, behavioural and physical variables, which is consistent with the proposed aetiological model. The next study will explore posttraumatic variables as outcome measures, with consideration given to the potential role of these variables in posttraumatic adjustment pathways.

This study has been previously presented in part, and referenced contributions by the author of this thesis are underlined:

Holmes, G.E., Williams, C.L., & Haines, J. (1998b, April). *Psychophysiological responses to personalized posttraumatic imagery following motor vehicle accidents.* Paper presented at the 25th Experimental Psychology Conference, Hobart, Australia.

Holmes, G.E., Williams, C.L., & Haines, J. (1998c, September). *Psychophysiological responses to personalized posttraumatic imagery following road trauma: A comparison of Posttraumatic Stress Disorder and Acute Stress Disorder.* Paper presented at the 9th World Congress on Psychophysiology, Sicily, Italy.

Holmes, G.E., Williams, C.L., & Haines, J. (1998d, October). *Dissociative phenomena in response to motor vehicle accident trauma.* Paper presented at the 2nd World Congress on Stress, Melbourne, Australia.

Holmes, G.E., Williams, C.L., & Haines, J. (1998e). Psychophysiological responses to personalized posttraumatic imagery following motor vehicle accidents. *Australian Journal of Psychology, 50,* 53-54.

Holmes, G.E., Williams, C.L., & Haines, J. (1998f). Psychophysiological responses to personalized posttraumatic imagery following road trauma: A comparison of Posttraumatic Stress Disorder and Acute Stress Disorder. *International Journal of Psychophysiology, 30,* 87.

Holmes, G.E., Williams, C.L., & Haines, J. (2000, September). *Psychophysiological and psychological responses to personalized posttraumatic imagery following motor vehicle accidents.* Paper presented to the Tasmanian Branch of the Australasian Society for Traumatic Stress Studies (ASTSS) in Hobart, Australia.

O'Donnell*, G.E.H., Williams, C.L., & Haines, J. (2001b). Posttraumatic psychophysiological and psychological responses to four stage guided imagery. *Manuscript submitted for review.*

*Note: Author's name changed from Holmes to O'Donnell during the course of this project due to marriage.

CHAPTER EIGHT

STUDY FOUR:

BIOPSYCHOSOCIAL OUTCOMES ASSOCIATED WITH POSTTRAUMATIC STRESS DISORDERS

8.1 Introduction

The human capacity to survive and adapt in response to trauma exposure has been contrasted with the human potential for chronic biological, psychological, occupational and social damage resulting from such experiences (e.g., van der Kolk & McFarlane, 1996). The proposal that posttraumatic responses may be characterized by an outcome continuum from the negative outcome of the development of chronic posttraumatic psychopathology to the other extreme of life changing personal growth has been widely supported by previous literature (e.g., Haines, Williams, Holmes, Wells, et al., 2001; Lyons, 1991; McFarlane & Yehuda, 1996; Valent, 1999). Although it is clear that trauma exposure results in extreme variance in biopsychosocial outcomes between individuals, it is not clear why this is so. By using the diagnostic framework of the posttraumatic psychological response definitions, the objective of this study is to explore in detail biopsychosocial outcomes associated with posttraumatic diagnoses. This objective was proposed to compare outcomes associated with the PTSD, ASD and subclinical groups, and explore if the outcomes associated with ASD are reflective of adaptive or disordered adjustment. The influence of posttraumatic variables on the differential development of posttraumatic responses will also be the subject of consideration.

8.1.1 Posttraumatic psychopathology

In terms of psychopathological outcomes, in addition to the diagnoses of PTSD and ASD, the comorbid development of other psychiatric diagnoses may occur. As previously discussed, psychopathology profiles of PTSD have indicated

high rates of comorbidity with depression, somatoform disorders, panic attacks and other anxiety disorders, substance-related disorders, personality disorders, brief psychoses, and dissociative disorders (e.g., Birmes, Arrieu, Payen, Warner, & Schmitt, 1999; Blanchard, Buckley, et al., 1998; Blanchard, Hickling, Taylor, & Loos, 1995; Bremner, 1999a; Breslau et al., 1991; Bryant, 1998; Deering et al., 1996; Everly, 1993; Koch & Taylor, 1995; O'Brien, 1998; Orsillo et al., 1998; Roemer et al., 1998; Shalev, 2000; Tryon, 1998). Measures of comorbidity have been proposed to identify the full spectrum of posttraumatic psychopathology, rather than the assessment ceasing solely with a posttraumatic diagnostic category, such as PTSD or ASD (e.g., Wilson & Keane, 1997).

Comorbidity has been comprehensively assessed using state-trait inventories, and general symptom assessment tools (e.g., Koretzky & Peck, 1990; Perrin, Van-Hasselt, & Hersen, 1997). In addition to the assessment of posttraumatic symptoms, these assessment instruments in combination have been proposed to provide expanded profiles of comorbidity, including personality factors that may be integral in the diagnosis, assessment and treatment of trauma survivors (e.g., Wilson & Keane, 1997).

8.1.2 ASD: Disorder versus adaptation

The development of the ASD diagnosis has been reported to have arisen out of an articulated need to describe distressing symptoms experienced during or soon after trauma exposure for psycholegal purposes (e.g., Bryant & Harvey, 2000a). The fact that ASD symptoms by definition have been described as short term indicates that individuals with this diagnosis may have protective psychological characteristics which mediate a more efficient recovery from initial

distress than do individuals with PTSD (e.g., Bryant & Harvey, 2000a), as opposed to the theoretical framework that ASD is a predictor of PTSD. In addition, the fact that such a short term diagnosis is considered a 'disorder' may be challenged by the proposal that individuals who develop ASD, without progressing to PTSD, experience an adaptive response for the longer term, and that ASD is not a 'disorder' at all. This proposition was supported by Koopman, Classen, Cardena, and Spiegel's (1995) review of studies on ASD, which stated that ASD may represent a form of psychological adaptation, protecting an individual from painful traumatic thoughts and feelings and allowing the individual to continue functioning. This proposition has been complicated by suggestions that ASD, or subsets of ASD symptoms such as dissociation, have been strongly related to the development of PTSD (Bryant, Harvey, Dang, Sackville, & Basten, 1998; Classen et al., 1998; Harvey & Bryant, 1998). As previously indicated, this series of studies has focused on the ASD subgroup who had not progressed to PTSD and, therefore, may represent the hypothesized adaptive nature of ASD. As stated by Wakefield (1997), the understanding of pathways that lead to avoidance of disorder in challenging environments has been considered of equal interest as studying the pathways to disorder.

8.1.3 Biopsychosocial outcomes

Biopsychosocial approaches to understanding the way individuals react to, and are affected by, traumatic experiences are based on the theoretical framework of interactions between the individual, family, community, social institutions, culture and the environment (e.g., Dilts, 2001; Ingram & Price, 2001; Saleeby, 2001). It has been suggested that consideration of the biological, psychological and

social outcomes of trauma exposure facilitates integrated needs assessments, in terms of intervention and treatment (e.g., Huyse et al., 2001).

Central to the definition of the diagnostic entities of PTSD and ASD is the evaluation of impairment of social and occupational functioning (APA, 1994). Impairments may be measured in terms of behavioural outcomes, such as inability to work or participate in social events; cognitive-affective outcomes, such as perceived dissatisfaction with different areas of one's life; and biological outcomes such as increased arousal impacting on functioning (e.g., APA, 1994; Buckley, 2000). Biopsychosocial measures, particularly those assessing psychosocial adjustment and perceived quality of life, have been reported to be strongly correlated with extent of PTSD symptoms (e.g., Buckley, 2000; Cordova et al., 1995; Hickling, Blanchard, Mundy, & Veazey, 1999; Zatzick et al., 1997). Biopsychosocial outcomes have been defined by a broad range of constructs, most of which affect multiple systems (e.g., Waysman et al., 2001). For example, situational anxiety and addictive behaviour may affect cognitive, behavioural and biological outcomes. A selection of biopsychosocial outcome measures was used in this study to investigate between group differences in a broad range of areas. Potential positive and negative influences of these biopsychosocial variables in the recovery environment will be considered as proposed by the integrated aetiological model.

Assessment tools, such as quality of life inventories, have been proposed to provide a more complete view of mental health status and adjustment than symptom-specific tools (e.g., Frisch, 1999; Landeen, Pawlick, Woodside, Kirkpatrick, & Byrne, 2000). This proposal has been based on the knowledge that the impact of illness on satisfaction with areas of life such as work, family

relationships and recreation differs between individuals (e.g., Helene & Ford, 2000), and the whole person, rather than the illness in isolation, requires consideration during assessment and treatment (e.g., Deahl et al., 2001). Quality of life measures offer valuable information for treatment planning and outcome assessments, and have been recommended to be used to compliment symptom-oriented psychological assessment tools (e.g., Frisch, 1999; Frisch, Cornell, Villanueva, & Retzlaff, 1992).

Quality of life measures have supported various investigations of posttraumatic influences on diagnostic outcome. For example, posttraumatic resource loss and role stress have been associated with severity of posttraumatic stress symptoms using these assessment tools (e.g., Hobfoll, 1988; Norris & Uhl, 1993; Thompson et al., 1998). In addition, issues such as life changes resulting from physical injury and chronic pain occurring directly as a result of an MVA have been related to severity of posttraumatic symptoms (e.g., Buckley, 2000). In a study of the psychosocial effects of exposure to an MVA, Hickling, Blanchard, Mundy, and colleague (1999) found that individuals diagnosed with PTSD reported significantly reduced quality of life and ability to attract and access social support than individuals without PTSD. Given that PTSD by definition is associated with longer term occupational and social impairment than ASD, it was perhaps obvious to speculate that the perceived quality of life of individuals diagnosed with PTSD would be significantly more negative in a broad range of life areas than the perceptions of individuals diagnosed with ASD. However, it may be considered less obvious to speculate that ASD, a label with psychiatric disorder status, may in fact predict positive posttraumatic outcomes in the longer term.

8.1.4 Aims and hypotheses

The aims of this study were to examine the nature of posttraumatic symptoms in individuals diagnosed with PTSD, ASD and subclinical categories of posttraumatic response; and to evaluate using multiple assessment tools the psychological impact of MVA trauma on resultant biopsychosocial functioning. The study was designed to identify specific targets for assessment, diagnosis and treatment of posttraumatic responses following MVA trauma, and speculated that posttraumatic variables associated with diagnostic outcomes mediate the differential development of posttraumatic stress disorders. It was hypothesized that:

1. The PTSD group would be the only group to endorse a wide range of clinically significant posttraumatic symptoms and broader comorbid psychopathology.
2. The profile of the ASD group would reflect posttraumatic adjustment, akin to the subclinical group, demonstrated by positive biopsychosocial outcomes.
3. The PTSD group would report a broad range of negative biopsychosocial outcomes.

8.2 Method

8.2.1 Participants

The sample was comprised of the 83 participants described in detail in study two. All participants had been exposed to an MVA meeting the DSM-IV criteria for "trauma", and divided into the PTSD, ASD and subclinical response groups, as previously established.

8.2.2 Materials

Structured clinical interview. The PACI (Holmes, 1997b) was designed for this research, as detailed in study two, and was utilized to establish group membership.

Impact of Event Scale (Revised) [IES-R]. The IES-R (Weiss & Marmar, 1997) is a 22-item self-report measure used to assess current posttraumatic symptomatology. The original IES (Horowitz et al., 1979) has been widely used for over two decades as a self-report measure of current posttraumatic intrusion and avoidance symptoms. The IES-R extends the usefulness of the original IES by including hyperarousal items, and other adjustments to improve the applicability of the scale to DSM-IV criteria. The revised scale has three subscales relating to three posttraumatic symptom clusters: intrusive symptoms, avoidance symptoms and hyperarousal symptoms. The measure is used to assess posttraumatic symptoms experienced during the past seven days, including the day of testing. Participants are instructed to indicate how distressed they felt by specific posttraumatic symptoms during the past seven days using a rating scale.

The subscales have been reported as internally consistent, and the measure has been noted to have good test-retest reliability (e.g., Matorin & Lynn, 1998). The split half reliability coefficient has been documented to be .86, whereas that for test-retest reliability was stated as .87. It has been reported to have good sensitivity (.92) and adequate specificity (.62). The test was developed and revised using non-combat trauma samples. The maximum test score is 88, and maximum subscale scores are Intrusive (32), Avoidance (32) and Hyperarousal (24). The IES-R (Weiss & Marmar, 1997) is displayed in Appendix E-1.

Beck Anxiety Inventory (BAI). The BAI (Beck, Epstein, Brown, & Steer, 1988) is a widely used 21 item questionnaire designed to assess symptoms of anxiety during the past seven days. The questionnaire has four symptom cluster subscales relating to neurophysiological (maximum score 21), subjective (18), panic (12) and autonomic (12) symptoms of anxiety. Each symptom item is rated in severity from zero to three. The BAI is reported to have high internal consistency reliability (Cronbach coefficient alpha .92), high test-retest reliability (.75) and substantial validity (Beck et al., 1988). Total BAI scores of 0-7 have been described as minimal levels of anxiety, 8-15 mild, 16-25 moderate and 26-63 severe (Beck & Steer, 1993).

Beck Depression Inventory (BDI). The BDI (Beck & Steer, 1987) is a widely used 21 item questionnaire designed to assess symptoms of depression during the past seven days (e.g., Beck, Steer, & Brown, 1996). Each item comprises four statements that reflect symptom severity. The statements are scaled from zero (no disturbance) to three (maximal disturbance). The total score can range from zero to 63. The BDI is reportedly a valid and reliable measure (e.g., Wenninger & Ehlers, 1998). Beck and Steer (1987) reported test-retest reliability as .74 and internal consistency alpha coefficients of .76 - .96 (Beck & Steer, 1987). The questionnaire has two subscales relating to cognitive-affective (maximum score 39) and physiological (maximum score 24) symptoms of depression. Four categories of symptoms severity are prescribed: score 0-9 minimal, 10-16 mild, 17-29 moderate, and 30-63 severe depression. Total BDI scores in excess of 15 have been termed potential indicators of clinical depression (Beck & Steer, 1987).

Symptom Checklist 90-Revised (SCL-90-R). The SCL-90-R (Derogatis, 1983) is a widely used 90 item questionnaire designed to assess patterns of psychological symptoms experienced during the week before assessment, in psychiatric and medical patients and non-patient samples. The questionnaire identifies the presence of symptoms warranting consideration for clinical intervention (e.g., Thompson et al., 1998). Each of the 90 items is rated on a five point scale assessing the severity of distress associated with experiencing each symptom. The test consists of nine primary symptom subscales: Somatization, Obsessive-Compulsive, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation, and Psychoticism.

In addition, three global indices have been designed to provide single scores of the nature and extent of psychological symptoms. The Global Severity Index (GSI) has been designed to provided a single summary score of the current level of psychopathology. The Positive Symptom Distress Index (PSDI) has been designed to provide a measure of perceived distress separate from the number of items endorsed. The Positive Symptom Total (PST) has been designed to evaluate the extent of symptomatology by scoring the number of endorsed items. Internal consistency for the subscales has been reported to range from .77 to .90, suggesting that the symptom items reflect the underlying factor proposed to be measured by each subscale. Test-retest reliability has been reported to range from .80 to .90, indicating stability across time. Convergent and construct validation studies have demonstrated the SCL-90-R to be a good measure of current psychopathology (Derogatis, 1983). A GSI score or two subscale scores above a standard score of 63 has been described as an indication of a positive diagnosis or

clinical case. Standard score norms for non-patient adults were used to derive standard scores for this sample (Derogatis, 1983).

Personality Assessment Inventory [PAI] (Morey, 1991). The PAI is a 344 item self-report inventory, and is used to assess a wide range of clinical and personality variables. These variables have been incorporated in a diagnostic framework including validity scales to assess the accuracy of self-report responses. This inventory was selected in preference to other clinical personality assessment tools due to its comprehensive validity scales component, scale types directly relevant to the investigation, and the availability of recent norms based on a community sample of adults (N = 1000) for appropriate sample comparison. The PAI consists of:

1. Four validity scales: Inconsistency, Infrequency, Negative Impression and Positive Impression.
2. Eleven clinical scales: Somatic Complaints, Anxiety, Anxiety-Related Disorders, Depression, Mania, Paranoia, Schizophrenia, Borderline Features, Antisocial Features, Alcohol Problems and Drug Problems.
3. Five treatment consideration scales: Aggression, Suicidal Ideation, Stress, Nonsupport, and Treatment Rejection
4. Two interpersonal scales: Dominance and Warmth.

Each item is rated on a four choice scale (false, slightly true, mainly true and very true). Subscales also have been defined for detailed analysis of the constructs assessed by the scales. The PAI is designed to assess these variables without a specific time frame, and the respondent is asked to rate each item in terms of 'how true' each statement is in describing them. T score values greater than 50 have been described to lie above the mean, and scores above 70 have been

said to represent a pronounced deviation in the adult community-dwelling standardization sample norms, chosen as the most appropriate comparison group for this research. Internal consistency for the subscales have been reported to range from .81 to .86, suggesting that the items reflect the underlying factor proposed to be measured. Test-retest reliability reports have ranged from .71 to .94 suggesting that the test is stable across time. Morey (1991) reported that comprehensive validation studies have demonstrated that the PAI is a strong measure of clinical and personality variables.

Trauma Symptom Inventory (TSI). The TSI (Briere, 1995) is a 100-item self-report questionnaire used to assess posttraumatic symptoms experienced during the previous six months. The TSI is a self-report measure of posttraumatic stress and other psychological sequelae of traumatic events. The TSI was devised with consideration to the DSM-IV criteria for PTSD and ASD, and intra and interpersonal difficulties often associated with traumatic experiences.

The TSI is composed of three validity scales (Response Level, Atypical Response and Inconsistent Response) and ten clinical scales (Anxious Arousal, Depression, Anger/Irritability, Intrusive Experiences, Defensive Avoidance, Dissociation, Sexual Concerns, Dysfunctional Sexual Behaviour, Impaired Self-Reference and Tension Reduction Behaviour).

The ten clinical scales have been reported to be internally consistent (e.g., mean alpha coefficient .87 in a university sample), and the validity scales have been reported to allow the detection of various types of response inconsistencies and anomalies. T scores are used to interpret age and sex specific responses in comparison to "general population" normative data. T scores above 65 have been reported to be clinically significant. The ten clinical scales can be categorized into

four distress factors: dysphoric mood (Anxious Arousal, Depression, and Anger/Irritability), posttraumatic stress (Intrusive Experiences, Defensive Avoidance, and Dissociation), sexual difficulties (Sexual Concerns and Dysfunctional Sexual Behaviour), and self-dysfunction (Impaired Self-Reference and Tension Reduction Behaviour). The sexual difficulties category is most applicable to trauma relating to sexual maltreatment, and is least relevant to MVA trauma.

The Stimulus-Response Inventory of Driving Related Situations [SRI-DRS] (Holmes, 1995). This inventory was designed by the author for this study to investigate between group differences in predicted psychophysiological response to potentially anxiety provoking driving related situations. The format of the inventory was based on the Stimulus-Response Inventory of Anxiousness (Endler & Hunt, 1969), and modified to include only psychophysiologically relevant items and driving specific stimuli. The inventory consists of the following five potential driving situations:

1. You are driving where the accident occurred.
2. You are the passenger in a car and the driver is speeding at 140km/h.
3. You are driving when a child runs onto the road in front of you.
4. You are driving and hear a loud crash behind you.
5. You are driving along an unfamiliar road in heavy rain.

These items were designed to target situations that may provoke anxiety in an individual who has been involved in an MVA. They were created to target visual and auditory stimuli associated with potential driving-related stressors. Respondents were asked how they thought they would react if they were placed in each of the situations on the day of testing. Perceived psychophysiological

response to being placed in each situation was rated on a five point Likert scale from zero (no response) to four (maximum response) for the following psychophysiological dimensions: heart rate, muscle tension, perspiration, coldness/numbness of fingers, and rapidity of breathing. The total score for each psychophysiological dimension (maximum 20), and the total score for the inventory (maximum 100) were calculated to compare the intensity and modes of perceived psychophysiological response. The SRI-DRS (Holmes, 1995) is displayed in Appendix E-2.

Quality of Life Inventory [QOLI]. The QOLI (Frisch, 1992) is a 32 item brief measure of life satisfaction. It assesses the importance of, and satisfaction with, 16 areas of life as perceived by the respondent. Each of the items is rated on a scale from -3 to +3. Internal consistency reliability (coefficient alpha) has been reported to be 0.79. Test-retest reliability has been reported to be 0.73. Each life area is assessed, and an overall quality of life score is calculated. Scores of 0-36 are categorized as very low, 37-42 low, 43-57 average, and 58-77 high quality of life.

8.2.3 Procedure

Ethical approval was obtained prior to participant recruitment. Voluntary participants were recruited from the university and community of Tasmania, as previously described. Structured clinical interviews (PACI; Holmes, 1997b) were then conducted. Each participant was then assessed using the battery of psychometric tests.

8.2.4 Design and data analysis

A three group design was employed using the PTSD, ASD and subclinical group division based on posttraumatic diagnoses. Dependent variables were responses to the structured interview and self-report inventories. Structured interview data were presented using frequency and percentage conversions with one way ANOVA and chi square analyses. MANOVA, one way ANOVA and LSD post hoc analyses were used to investigate between group differences in the psychometric scale scores of the IES-R, BAI, BDI, SCL-90-R, TSI, PAI, SRI-DRS and QOLI. MANOVAs were employed due to their sensitivity not only to mean differences, but also to the direction and size of correlations among the dependent variables. Significance levels were set at .05 for all tests, with Bonferroni corrections applied to reduce the possibility of Type 1 errors resulting from multiple significance tests.

8.3 Results

8.3.1 Overview

The symptom profiles of each group, in terms of the DSM-IV (APA, 1994) criteria, were presented using data elicited from the structured interview. Current psychopathology group profiles were described using data obtained from the IES-R, BAI, BDI and SCL-90-R. Psychometric profiles of psychopathology and clinical personality variables were presented using data obtained from the PAI and TSI. The results obtained using these two measures were presented in an integrated section, as they offered complimentary measures of validity and clinical dimensions, and reflected longer term functioning than the other measures that

focused exclusively on the week prior to testing. Posttraumatic changes in biopsychosocial outcome measures were then presented.

8.3.2 DSM-IV criteria

The PACI (Holmes, 1997b) elicited data regarding each of the diagnostic criteria for PTSD and ASD in order to determine posttraumatic diagnostic status for group membership. This data has been chosen for presentation here, in order to provide a clinical picture of the nature and extent of the experience of each symptom type by each group, in addition to other outcome measures. As previously noted, a case by case presentation of symptom profiles is displayed in Appendix C-3. Table 17 displays the percentages and frequencies of each group meeting the DSM-IV (APA, 1994) criteria for each of the symptom types of ASD and PTSD. It was noted that none of the participants in the PTSD group met the criteria for a diagnosis of ASD prior to the development of PTSD due to the relatively low level of dissociative symptoms experienced by this group. All participants in the PTSD group did meet the ASD criteria C, D, and E during the first four weeks posttrauma. The data presented reflect that the subclinical group was generally not asymptomatic and is arguably best described as having experienced posttraumatic symptoms that did not meet the criteria for PTSD or ASD. It can be seen that higher percentages of the subclinical group reported experiencing various posttraumatic symptoms than the ASD group after the first month.

Chi square analyses demonstrated significant differences between the three groups on all criteria. In relation to the ASD symptoms, both the PTSD and subclinical groups differed significantly from the ASD group on all criteria. In

relation to the PTSD symptoms, both the ASD and subclinical groups differed significantly from the PTSD group on all but two criteria. Interestingly, the ASD group reported significantly greater endorsement of two of the avoidance and numbing criteria, inability to recall (C3) and detachment (C5), than the other groups, and these criteria have been proposed to be dissociative, rather than avoidant (e.g., van der Kolk, McFarlane, et al., 1996; van der Kolk, Pelcovitz, et al., 1996).

Table 17.

Frequency data with conversion into percentages and between group chi square comparisons for each of the symptom types of ASD and PTSD (N = 83).

ASD criterion (duration 2 days-4 weeks)		PTSD ($n = 30$)	ASD ($n = 24$)	Subclinical ($n = 29$)	χ^2 *$p < .05$
		Freq.(%)	*Freq.*(%)	*Freq.*(%)	($df = 2$)
B	**Dissociation**	0 (0)	24 (100)	0 (0)	49.9*
B1	Detachment	2 (13)	23 (96)	3 (10)	43.2*
B2	Reduced awareness	6 (20)	22 (92)	5 (17)	41.7*
B3	Derealization	4 (13)	22 (92)	4 (14)	40.8*
B4	Depersonalization	0 (0)	19 (79)	0 (0)	37.6*
B5	Dissociative amnesia	1 (3)	9 (38)	3 (10)	20.4*
C	**Reexperiencing**	30 (100)	24 (100)	20 (69)	28.3*
D	**Avoidance and numbing**	30 (100)	24 (100)	12 (41)	34.2*
E	**Increased arousal**	30 (100)	24 (100)	16 (55)	36.7*

(Table continues...)

Table 17 (continued...)

PTSD criterion (duration > one month)		PTSD (n = 30)	ASD (n = 24)	Subclinical (n = 29)	χ^2 *p < .05
		Freq.(%)	*Freq.*(%)	*Freq.*(%)	(df = 2)
B	**Reexperiencing**	30 (100)	4 (17)	11 (38)	35.6*
B1	Recollections	24 (80)	4 (17)	11 (38)	24.2*
B2	Dreams	20 (67)	2 (8)	8 (28)	18.5*
B3	Reliving	12 (40)	4 (17)	4 (14)	10.7*
B4	Exposure to cues	24 (80)	4 (17)	10 (35)	23.8*
B5	Physiological reactivity	24 (80)	2 (8)	10 (35)	28.9*
C	**Avoidance and numbing**	30 (100)	2 (8)	7 (24)	42.3*
C1	Thoughts/feelings	16 (53)	1 (4)	7 (24)	21.3*
C2	Activities/places/people	16 (53)	1 (4)	7 (24)	22.1*
C3	Inability to recall	9 (30)	9 (38)	0 (0)	10.2*
C4	Diminished interest	26 (87)	1 (4)	5 (17)	25.2*
C5	Detachment	16 (53)	20 (84)	2 (7)	11.5*
C6	Restricted affect	15 (50)	1 (4)	5 (17)	13.7*
C7	Foreshortened future	15 (50)	0 (0)	5 (17)	15.4*
D	**Increased arousal**	30 (100)	1 (4)	7 (24)	41.7*
D1	Sleeping difficulties	30 (100)	1 (4)	8 (28)	41.2*
D2	Anger/irritability	21 (70)	0 (0)	7 (24)	35.7*
D3	Concentration difficulties	19 (63)	1 (4)	5 (17)	21.2*
D4	Hypervigilance	18 (60)	0 (0)	7 (24)	15.7*
D5	Startle response	15 (50)	0 (0)	4 (14)	23.5*

Posttraumatic panic attacks were reported by 50% of the PTSD group, 4% of the ASD group and 14% of the subclinical group, with no participants reporting a pretrauma history of panic attacks. At least one posttraumatic Major Depressive Episode was reported by 50% of the PTSD group, 4% of the ASD group and 17% of the subclinical group, with all participants reporting that they had not experienced symptoms meeting the criteria for a Major Depressive Episode before the MVA. No participants reported symptoms meeting the criteria for a Manic Episode or Brief Psychotic Disorder with Marked Stressor. Ninety percent of the PTSD diagnoses were current and chronic. The remaining ten percent of PTSD diagnoses were current and acute. There were no PTSD diagnoses characterized by delayed onset, and no retrospective diagnoses of PTSD.

8.3.3 Current psychopathology

A selection of both trauma-specific and general psychopathology measures were utilized to assess the self-reported symptoms that the three groups experienced during the week prior to testing. These measures were selected to provide a clinical picture of the self-reported current psychological functioning of the three groups.

8.3.3.1 IES-R

A MANOVA indicated that there were significant multivariate group differences for the IES-R scales, *Rao's R* (6, 156) = 4.97, $p = .0001$. One-way ANOVAs and LSD post hoc analyses indicated that the PTSD group scores were significantly higher than the ASD group scores on the Intrusive (*Fisher LSD* = 14.32, $p < .05$); Avoidance (*Fisher LSD* = 9.83, $p < .05$); Hyperarousal (*Fisher*

LSD = 10.92, $p < .05$) scales; and the total IES-R score (*Fisher LSD* = 13.68, $p <$ .05). Table 18 displays the mean scores and standard deviations for each group on the IES-R scales.

Table 18.

Group means (standard deviations) and one-way ANOVA results for each subscale and total score on the IES-R (N = 83).

Subscale	Group								
	PTSD		ASD		Subclinical		One way ANOVA		
	M	*(SD)*	*M*	*(SD)*	*M*	*(SD)*	*F (2,80)*	*p*	Post hoc
Intrusive	15.10	(8.67)	9.92	(7.29)	4.48	(5.17)	16.02	<.0001	P>A>S
Avoidance	10.87	(7.82)	7.67	(6.91)	3.17	(4.70)	10.07	<.0001	P>A>S
Hyperarousal	9.83	(6.45)	7.13	(5.45)	2.93	(4.20)	11.92	<.0001	P>A>S
Total IES-R	35.80	(20.97)	24.71	(18.66)	10.59	(13.18)	14.68	<.0001	P>A>S

*All differences significant after Bonferroni correction (.05/4 = .0125).

8.3.3.2 BAI

A MANOVA indicated that there were significant multivariate group differences for the BAI subscales, *Rao's R* (8, 154) = 2.57, $p = .01$. Table 19 displays the mean scores, standard deviations and one way ANOVA results for each group on the subscales and total BAI.

One-way ANOVAs and LSD post hoc analyses indicated that the PTSD group scores were significantly higher than the ASD and subclinical group scores on the Neurophysiological (*Fisher LSD* = 3.68, $p < .05$); Subjective (*Fisher LSD* = 5.67, $p < .05$); and Autonomic (*Fisher LSD* = 8.36, $p < .05$) subscales. There were

no significant differences on the Panic subscale. The PTSD group scored significantly more highly on the BAI than the other groups (*Fisher LSD* = 7.68, $p <$ .05). The PTSD group score was reflective of moderate anxiety, whereas the other group scores reflected minimal levels of anxiety.

Table 19.

Group means (standard deviations) and one-way ANOVA results for each subscale and total score on the BAI (N = 83).

Subscale	Group					
	PTSD	ASD	Subclinical	One way ANOVA		
	M (SD)	*M (SD)*	*M (SD)*	*F (2,80)*	*p*	Post hoc
Neurophysiological	3.80 (3.42)	2.00 (3.49)	1.59 (1.94)	4.47	.01	P>A,S
Subjective	6.20 (3.54)	2.50 (3.51)	2.45 (2.82)	6.56	.002	P>A,S
Panic	1.60 (2.25)	0.75 (1.42)	0.62 (0.94)	3.01	.054	n.a.
Autonomic	4.83 (3.04)	1.50 (1.87)	1.62 (1.61)	9.37	.0002	P>A,S
Total BAI score	16.43(10.61)	6.75 (9.11)	6.28 (5.06)	8.22	.0006	P>A,S

*All differences significant after Bonferroni correction (.05/5 = .01).

8.3.3.3 BDI

A MANOVA found significant multivariate group differences for the BDI scales, *Rao's R* (4, 158) = 4.80, p = .001. Table 20 displays the mean scores, standard deviations and one-way ANOVA results for each group on the BDI.

One-way ANOVAs and LSD post hoc analyses indicated that the PTSD group scores were significantly higher than the ASD and subclinical group scores on the Cognitive-affective (*Fisher LSD* = 6.68, $p <$ .05) and Physiological (*Fisher*

LSD = 7.76, $p < .05$) BDI subscales, and the BDI total scale scores (*Fisher LSD* = 8.76, $p < .05$). The PTSD group score was reflective of a moderate level of depression, and the ASD and subclinical group scores reflected minimal symptoms of depression.

Table 20.

Group means (standard deviations) and one-way ANOVA results for each subscale and total score on the BDI (N = 83).

Subscale	Group					
	PTSD	ASD	Subclinical	One way ANOVA		
	M (*SD*)	*M* (*SD*)	*M* (*SD*)	*F* (2,80)	*p*	Post hoc
Cognitive-affective	10.33 (9.09)	3.53 (3.47)	4.94 (5.05)	7.78	<.0001	P>A,S
Physiological	7.07 (4.67)	3.06 (2.73)	3.81 (3.16)	8.77	<.0001	P>A,S
Total BDI score	17.40(12.69)	6.59 (5.68)	8.75 (7.15)	9.90	<.0001	P>A,S

*All differences significant after Bonferroni correction (.05/3 = .017).

8.3.3.4 SCL-90-R

A MANOVA indicated that there were significant multivariate group differences for the SCL-90-R scales, *Rao's R* (18, 144) = 2.75, $p = .0004$. One-way ANOVAs and LSD post hoc analyses with Bonferroni correction indicated that the PTSD group scores were significantly higher than the ASD and subclinical group scores on the Somatization (*Fisher LSD* = 11.87, $p < .05$), Obsessive-compulsive (*Fisher LSD* = 10.27, $p < .05$), Depression (*Fisher LSD* = 9.57, $p < .05$), and Anxiety (*Fisher LSD* = 8.72, $p < .05$) subscales, and the GSI (*Fisher LSD* = 11.77, $p < .05$), PSDI (*Fisher LSD* = 15.45, $p < .05$), and PST

(*Fisher LSD* = 9.88, $p < .05$). The mean scores of the PTSD group on these dimensions exceeded 63, fulfilling the criterion for clinical caseness. There were no significant differences on the Interpersonal sensitivity, Hostility, Phobia, Paranoia and Psychosis scales after Bonferroni correction. The mean score of the PTSD group on the Interpersonal sensitivity scale fulfilled the criterion for a clinical case. None of the ASD and subclinical group scores reached the clinical criterion of 63. Table 21 displays the mean standard scores and standard deviations for each group on the subscales and SCL-90-R total score indices.

Table 21.

Group means (standard deviations) and one-way ANOVA results for each subscale and total score index on the SCL-90-R (N = 83).

Subscale	Group								
	PTSD		ASD		Subclinical		One way ANOVA		
	M	*(SD)*	*M*	*(SD)*	*M*	*(SD)*	*F (2,80)*	*p*	Post hoc
Somatization	65.33	(8.62)	52.67	(12.14)	54.10	(10.28)	12.85	<.0001*	P>A,S
Obsessive-C	66.63	(8.82)	57.50	(10.84)	55.21	(9.32)	11.56	<.0001*	P>A,S
Interpersonal-S	64.47	(11.09)	56.50	(9.06)	56.79	(12.85)	4.64	.01	P>A,S
Depression	66.50	(7.24)	59.33	(8.73)	55.86	(10.95)	10.45	<.0001*	P>A,S
Anxiety	63.27	(10.16)	52.96	(12.13)	52.24	(10.84)	9.11	.0003*	P>A,S
Hostility	57.00	(9.26)	52.58	(11.99)	51.03	(9.03)	2.78	.07	n.a.
Phobia	58.10	(11.59)	53.92	(10.71)	49.62	(7.22)	5.30	.007	n.a.
Paranoia	58.30	(11.05)	53.54	(9.63)	52.28	(9.19)	2.94	.06	n.a.

(Table continues...)

Table 21 (continued...)

Subscale	Group						One way ANOVA		
	PTSD		ASD		Subclinical				
	M	*(SD)*	*M*	*(SD)*	*M*	*(SD)*	*F (2,80)*	*p*	Post hoc
Psychosis	62.03	(10.43)	56.88	(10.65)	56.03	(10.96)	2.69	.07	n.a.
GSI	67.20	(7.12)	57.00	(10.56)	55.52	(11.38)	12.28	<.0001*	P>A,S
PSDI	64.50	(8.23)	52.75	(8.38)	54.48	(8.36)	16.36	<.0001*	P>A,S
PST	64.47	(6.95)	57.46	(10.12)	54.17	(9.32)	10.52	<.0001*	P>A,S

* Significant after Bonferroni correction (.05/12 = .004).

8.3.4 Psychometric profiles of psychopathology and personality

The PAI and TSI were used to examine posttraumatic symptom and personality profiles.

8.3.4.1 PAI and TSI validity scales

A MANOVA indicated that the three groups showed no significant multivariate differences for the PAI validity scales, *Rao's R* (8,154) = 1.75, p = .09. A MANOVA indicated significant multivariate differences for the TSI validity scales, *Rao's R* (6,156) = 5.02, p = .0001. One-way ANOVAs and LSD post hoc analyses indicated that the PTSD group score was significantly higher than the scores of the other two groups on the Atypical Responses scale (*Fisher LSD* = 8.44, $p < .05$), and the subclinical group score was significantly higher than the other groups on the Response Level (*Fisher LSD* = 6.25, $p < .05$) scale. However, the scores for the three groups on all PAI and TSI validity scales were within acceptable limits, indicating valid assessments. Table 22 displays the means,

standard deviation and breakdown one-way ANOVA results for the PAI and TSI validity scales.

Table 22.

Means (standard deviations) and one way ANOVA results for the PAI and TSI validity scales (N = 83).

Scale	Group							
	PTSD		ASD		Subclinical		One way ANOVA	
	M	*(SD)*	*M*	*(SD)*	*M*	*(SD)*	*F (2,80)*	*p*
PAI								
Inconsistency	42.91	(7.61)	41.31	(7.01)	42.71	(8.71)	n.a.	
Infrequency	54.12	(8.92)	53.81	(7.02)	52.52	(10.82)	n.a.	
Negative Impression	60.43	(11.33)	51.42	(13.13)	49.83	(7.43)	n.a.	
Positive Impression	46.62	(11.92)	48.53	(10.02)	50.43	(9.13)	n.a.	
TSI								
Atypical Response	57.07	(12.51)	49.13	(5.62)	48.38	(4.99)	9.00 (.0003)**	
Response Level	43.63	(5.08)	44.88	(5.40)	49.83	(9.39)	6.38 (.003)**	
Inconsistency	44.17	(5.64)	43.58	(5.70)	41.55	(4.99)	1.84 (.16)	

** One way ANOVA significance levels after Bonferroni correction:

[TSI validity] .05/3 = .02

8.3.4.2 PAI and TSI clinical scales

A MANOVA indicated that the three groups showed significant multivariate differences for the PAI clinical scales, *Rao's R* (22,140) = 2.85, p = .0001; and the TSI clinical scales, *Rao's R* (20,142) = 2.39, p = .002. Table 23

presents the means, standard deviations, *F* values and associated *p* values for the PAI and TSI clinical scales.

Table 23.

Means (standard deviations) and one way ANOVA results for the PAI and TSI clinical scales and subscales (N = 83).

Scale	Group				
	PTSD	ASD	Subclinical	One way ANOVA	
	M (SD)	*M (SD)*	*M (SD)*	*F (2,80)*	*p*
Somatic C.	67.21 (11.71)	51.33 (8.92)	55.62 (12.33)	15.00	(<.0001)**
Anxiety	60.42 (11.32)	51.42 (13.12)	49.81 (9.92)	7.31	(.001) **
Anxiety D.	62.23 (14.21)	50.81 (13.03)	48.12 (10.82)	10.62	(<.0001)**
Depression	65.21 (14.21)	52.53 (9.61)	53.42 (13.42)	8.73	(.0004)**
Schizophrenia	58.82 (12.51)	49.52 (8.23)	49.81 (8.22)	6.62	(.002) **
Drug Problems	56.73 (10.32)	52.31 (7.83)	49.11 (8.22)	5.31	(.007) *
Alcohol P.	54.21 (16.00)	52.82 (10.12)	49.42 (7.82)	.73	(.52)
Mania	51.42 (10.91)	51.43 (12.61)	48.03 (8.22)	1.01	(.38)
Paranoia	51.33 (9.52)	50.32 (9.51)	48.32 (9.71)	.82	(.47)
Borderline F.	59.01 (11.73)	54.42 (12.10)	53.34 (10.12)	2.12	(.13)
Affective I.	56.92 (11.73)	54.02 (13.41)	52.22 (11.02)	1.21	(.32)
Identity P.	58.31 (14.94)	55.91 (11.32)	52.42 (8.74)	1.83	(.17)
Negative R.	60.11 (10.23)	50.52 (11.33)	52.02 (11.23)	6.42	(.003) **
Self-Harm	49.82 (12.33)	49.83 (9.32)	53.52 (11.43)	1.02	(.36)

(Table continues...)

Table 23 (continued...)

Scale	Group						One way ANOVA	
	PTSD		ASD		Subclinical			
	M	*(SD)*	*M*	*(SD)*	*M*	*(SD)*	*F (2,80)*	*p*
Antisocial F.	50.21	(10.54)	52.81	(9.42)	52.82	(9.74)	.71	(.52)
Antisocial B.	50.31	(10.62)	49.42	(7.41)	51.71	(10.10)	.43	(.68)
Egocentricity	48.22	(7.00)	55.73	(10.62)	51.72	(8.60)	4.92	(.010) *
Stimulus-S	51.11	(12.51)	52.62	(13.91)	53.70	(10.84)	.34	(.72)
Anxious A.	60.63	(10.32)	51.58	(9.94)	49.62	(8.30)	11.04	(<.0001)**
Depression	56.27	(10.33)	52.21	(8.18)	49.93	(10.42)	3.16	(.04) *
Anger & I.	57.03	(11.38)	52.17	(8.62)	48.48	(8.65)	5.74	(.004) **
Intrusive E.	60.20	(11.46)	51.42	(8.80)	50.06	(7.66)	9.73	(<.0001)**
Defensive A.	59.00	(11.92)	52.37	(9.51)	48.90	(7.67)	7.91	(.0007)**
Dissociation	61.33	(10.63)	54.50	(8.41)	50.06	(7.21)	11.94	(<.0001)**
Sexual C.	53.70	(11.05)	50.83	(9.26)	50.27	(9.74)	.96	(.39)
Dysfunctional S.	50.43	(11.81)	55.63	(16.43)	51.79	(11.52)	1.07	(.35)
Impaired S.R.	55.83	(10.47)	53.46	(8.41)	50.06	(8.22)	2.94	(.06)
Tension R.B.	55.67	(13.26)	55.63	(12.95)	50.75	(11.56)	1.42	(.25)

* Significant at .05 level.

** One way ANOVA significance levels after Bonferroni correction:

[PAI clinical] .05/11 = .005

[TSI clinical] .05/10 = .005

One way ANOVA and LSD post hoc procedures indicated that the PTSD group scored significantly more highly than the other groups on the following PAI clinical scales: Somatic Complaints (*Fisher LSD* = 13.35, $p < .05$), Anxiety (*Fisher LSD* = 6.67, $p < .05$), Anxiety-Related Disorders (*Fisher LSD* = 10.55, $p < .05$), Depression (*Fisher LSD* = 7.99, $p < .05$), Schizophrenia (nonpsychotic symptoms) (*Fisher LSD* = 5.68, $p < .05$), and Drug Problems (*Fisher LSD* = 5.25, $p < .05$). Following Bonferroni correction, the main effect for Drug Problems was no longer significant. The ASD and subclinical group scores were not significantly different. There were no significant between group differences on the Alcohol Problems scale, with alcohol use reported within the average range by all groups. There were also no significant between group differences on the Mania or Paranoia scales. Group means were within the average range on these two scales.

The PAI Borderline and Antisocial Features scales assessed the characteristics of borderline and antisocial personality profiles. There were no significant main effects for either scale. However, there was a significant main effect for the Negative Relationships subscale of the Borderline Features scale, with post hoc analyses showing that the PTSD group scored significantly higher than the ASD and subclinical groups on this subscale (*Fisher LSD* = 5.88, $p < .05$). The ASD and subclinical group means were not significantly different.

In addition, there was a significant main effect for the Egocentricity subscale of the Antisocial Features scale, with post hoc analyses showing that the ASD group scored significantly higher than the subclinical and PTSD groups on this subscale (*Fisher LSD* = 3.98, $p < .05$). The PTSD and subclinical group means were not significantly different. It should be emphasized that the mean score of the ASD group was still within the average range, and was not indicative of antisocial

psychopathology. Following Bonferroni correction, the main effect for Egocentricity was no longer significant. Given that the statistical procedures used are stringent for this clinical sample, the finding has been noted for exploratory purposes. All other borderline and antisocial feature subscale scores were not significantly different between groups, and all scores were within the average range.

One way ANOVA and LSD post hoc procedures indicated that the PTSD group scored significantly more highly than the other groups on the following TSI clinical scales from the dysphoric mood and posttraumatic stress categorizations of the TSI: Anxious Arousal (*Fisher LSD* = 10.68, $p < .05$), Intrusive Experiences (*Fisher LSD* = 8.78, $p < .05$), Defensive Avoidance (*Fisher LSD* = 7.45, $p < .05$) and Dissociation (*Fisher LSD* = 11.26, $p < .05$). The ASD and subclinical group scores were not significantly different on these scales. The PTSD group scored significantly more highly than the subclinical group on the dysphoric mood category dimensions, the Depression (*Fisher LSD* = 2.99, $p < .05$) and Anger/Irritability (*Fisher LSD* = 5.35, $p < .05$) scales, of the TSI. The PTSD group scores on the Anxious Arousal, Intrusive Experiences and Defensive Avoidance subscales were above the clinical cut-off. No between group differences were found on the Sexual Concerns, Dysfunctional Sexual Behaviour, Impaired Self Reference and Tension Reduction Behaviour scales, that comprise the sexual difficulties and self-dysfunction categories of distress.

8.3.4.3 PAI treatment consideration and interpersonal scales

Table 24 presents the mean scores and standard deviations for the five treatment consideration scales and the two interpersonal scales.

Table 24.

Group means (standard deviations) for the PAI Treatment Consideration and Interpersonal scales (N = 83).

Scale	Group		
	PTSD	ASD	Subclinical
	M (SD)	*M (SD)*	*M (SD)*
Treatment Consideration Scales			
Aggression	48.1 (10.0)	49.8 (11.6)	47.6 (10.1)
Suicidal Ideation	57.1 (14.5)	51.3 (8.7)	56.6 (16.2)
Stress	58.2 (11.7)	52.9 (6.3)	53.9 (10.6)
Nonsupport	53.0 (13.4)	47.1 (8.3)	49.0 (11.8)
Treatment Rejection	47.1 (10.0)	53.1 (9.6)	47.1 (10.0)
Interpersonal Scales			
Dominance	49.7 (10.6)	53.0 (12.6)	48.2 (10.1)
Warmth	47.6 (12.4)	55.0 (7.0)	50.8 (10.5)

The treatment consideration scales were designed to provide information about issues which may complicate treatment programs. A MANOVA indicated that the three groups showed no significant multivariate differences for the treatment consideration scales, *Rao's R* (10,152) = 1.19, $p = .30$. All group means were within the average range.

The interpersonal scales were designed to assess the extent of dominance and warmth exerted in interpersonal relationships. Given that there were only two interpersonal scales, one way ANOVA were conducted for these two scales. One way ANOVA indicated that there was a significant main effect for the Warmth

scale, F (2,80) = 3.4, $p < .05$. The Warmth scale assessed the extent to which a person is empathic and engaging in interpersonal relationships. Post-hoc analyses showed that the ASD group scored significantly higher than the PTSD group on this scale (*Fisher LSD* = 3.26, $p < .05$). All group means were within the average range. There was no significant main effect for the Dominance scale, and all group means were within the average range.

8.3.5 Biopsychosocial outcome measures

A selection of biopsychosocial outcome measures was chosen to investigate between group differences in road travel-specific variables, addictive behaviours and quality of life perceptions. These variables were proposed to represent cognitive-affective and behavioural outcome measures, descriptive of positive and negative lifestyle changes associated with MVA trauma.

8.3.5.1 Driving and traveling by road: Posttraumatic changes in attitudes and behaviour

There were no significant between group differences in reported changes in driving behaviour and attitude to traveling by road, χ^2 (4, N = 83) = 7.47, $p > .05$. The majority of the participants in each of the three groups reported that the MVA had negatively affected the way they felt about driving and traveling by road (PTSD 75%, ASD 70%, subclinical 73%); and this was also reflected in reported changes in behaviour associated with traveling by road, such as avoidance (PTSD 70%, ASD 65%, subclinical 69%). The cognitive and behavioural effects were quantitatively reported as more enduring for the PTSD group, with the other groups reporting these effects predominantly in the first weeks after the trauma and

diminishing with time, as determined by responses to the structured interview. A minority of participants reported that the MVA had positively affected the way they felt about driving and traveling by road, and had positive behavioural results, such as being more cautious when traveling by road and adhering to speed limits (PTSD 8%, ASD 15%, subclinical 12%). A small number of participants reported no change in attitude or behaviour associated with road travel (PTSD 3%, ASD 10%, subclinical 8%).

A MANOVA indicated that there were significant multivariate group differences for the SRI-DRS scales, *Rao's R* (8, 154) = 5.28, $p = .0001$, indicating significant differences in perceived psychophysiological response to driving-related situations. The results are described in terms of each group's prediction of their response to these potentially anxiety provoking situations. Significant main effects were found for predicted perspiration response, $F(2,80) = 5.27$, $p < .05$, and total score, $F(2,80) = 8.27$, $p < .05$. These differences were found to be significant after Bonferroni correction (.05/6 = .008). The means and standard deviations for each predicted psychophysiological response and the total SRI-DRS (Holmes, 1995) scores for each group are presented in Table 25. Post hoc analyses demonstrated that the PTSD and ASD groups predicted a significantly greater perspiration response to the potentially anxiety provoking situations than the subclinical group (*Fisher LSD* = 2.68, $p < .05$). In addition, the total score of the PTSD and ASD groups on the SRI-DRS (Holmes, 1995) was significantly higher than the subclinical group (*Fisher LSD* = 3.77, $p < .05$), reflecting that the PTSD and ASD groups predicted greater psychophysiological arousal in response to the potentially anxiety provoking driving related situations.

Table 25.

Group mean scores (standard deviations) for each psychophysiological response category of the SRI-DRS (Holmes, 1995) (N = 83).

Psychophysiological response	Group					
	PTSD		ASD		Subclinical	
	M	*(SD)*	*M*	*(SD)*	*M*	*(SD)*
Heart beats faster	13.61	(4.00)	13.84	(3.61)	11.14	(4.31)
Muscles become tense	13.42	(4.71)	12.64	(4.12)	10.31	(4.03)
Perspire	8.22	(5.33)	6.81	(4.34)	4.03	(3.74)
Fingers feel cold or numb	4.21	(5.54)	5.33	(4.93)	2.22	(3.60)
Breathing becomes rapid	9.73	(5.02)	12.62	(10.00)	7.70	(4.22)
Total score	49.10	(18.70)	49.43	(17.62)	35.24	(15.91)

Trends for main effects were found for predicted heart rate, F (2,80) = 2.71, p = .07; muscle tension, F (2,80) = 2.90, p =.06; and rapid breathing, F (2,80) = 2.50, p = .09. Post hoc analyses demonstrated that the PTSD and ASD groups predicted a significantly greater heart rate response than the subclinical group (*Fisher LSD* = 2.54, $p < .05$), the PTSD group predicted significantly greater muscle tension than the subclinical group (*Fisher LSD* = 2.78, $p < .05$), and the ASD group predicted significantly more rapid breathing than the subclinical group (*Fisher LSD* = 2.43, $p < .05$) in response to the potentially anxiety provoking driving related situations. There were no other between group differences in predicted psychophysiological response measured by the SRI-DRS (Holmes, 1995).

8.3.5.2 Addictive behaviour

There were no significant group differences in reported posttraumatic changes in potentially addictive behaviours that may be used as coping strategies (alcohol consumption, χ^2 (6, N = 83) = 7.48, p > .05; smoking tobacco, χ^2 (6, N = 83) = 0.66, p > .05; other drug use, χ^2 (6, N = 83) = 3.87, p > .05; and gambling, χ^2 (6, N = 83) = 6.87, p > .05). It should be noted that the "other drugs" category referred to recreational drug use, not prescription medication. Table 26 displays the percentage of each group reporting changes in alcohol consumption, smoking, other drug use and gambling.

Table 26.

Frequency data with conversion into percentages for each group reporting perceived posttraumatic changes in potentially addictive behaviours (N = 83).

Subscale		Group		
		PTSD	ASD	Subclinical
		Freq.(%)	Freq.(%)	Freq.(%)
Alcohol consumption	Nil	5 (17)	4 (17)	5 (17)
	No change	18 (61)	15 (63)	19 (65)
	Decreased	3 (10)	2 (8)	2 (8)
	Increased	4 (12)	3 (12)	3 (10)
Smoking tobacco	Nil	20 (68)	16 (68)	20 (69)
	No change	5 (17)	4 (17)	5 (17)
	Decreased	1 (3)	1 (4)	1 (3)
	Increased	4 (14)	3 (11)	3 (10)

(Table continues...)

Table 26 (continued...)

Subscale		Group		
		PTSD	ASD	Subclinical
		Freq.(%)	Freq.(%)	Freq.(%)
Other recreational drug use	Nil	24 (82)	20 (82)	24 (81)
	No change	3 (9)	3 (11)	3 (11)
	Decreased	0 (0)	0 (0)	0 (0)
	Increased	3 (9)	1 (7)	2 (8)
Gambling	Nil	22 (72)	17 (72)	21 (72)
	No change	7 (24)	6 (25)	7 (23)
	Decreased	0 (0)	0 (0)	0 (0)
	Increased	1 (4)	1 (3)	1 (5)

Table 26 shows that most participants reported no perceived change in alcohol consumption, were non-smokers, did not report engaging in other recreational drug use, or gambling. Less than 15% of participants reported perceived changes in these behaviours following the MVA.

8.3.5.3 Posttraumatic perceived quality of life

MANOVA demonstrated that there were significant between group differences in mean scores on the QOLI, *Rao's R* (32, 126) = 3.01, p <.0001. One way ANOVAs with Bonferroni correction (p = .05/16 = .003) were used to investigate the nature of these differences. The PTSD group perceived overall quality of life, as measured by QOLI T-scores, to be significantly poorer than the ASD and subclinical groups, F (2,80) = 31.66, p < .0001 (*Fisher LSD* = 21.72, p <

.05; PTSD M = 38.30, SD = 6.58; ASD M = 54.79, SD = 9.73; subclinical M = 55.21, SD = 10.90). Group means, standard deviations and one way ANOVA results for each subscale of the QOLI are shown in Table 27.

The analyses demonstrated that the PTSD group perceived quality of life in the following areas to be significantly poorer than the ASD and subclinical groups: Health (*Fisher LSD* = 23.32, $p < .05$), Self esteem (*Fisher LSD* = 15.45, $p < .05$), Money (*Fisher LSD* = 7.66, $p < .05$), Work (*Fisher LSD* = 16.78, $p < .05$), Play (*Fisher LSD* = 35.41, $p < .05$), Learning (*Fisher LSD* = 12.12, $p < .05$), Love (*Fisher LSD* = 7.62, $p < .05$), Friends (*Fisher LSD* = 12.41, $p < .05$) and Relatives (*Fisher LSD* = 12.32, $p < .05$).

Trends were was also found for the PTSD group to rate perceived quality of life in the following areas to be poorer than the ASD and subclinical groups: Goals and values, Helping, Home and Community. However, these trends were not significant after Bonferroni correction. No significant between group differences were found on the Creativity, Children, and Neighbourhood subscales.

Table 27.

Group means (standard deviations) and one-way ANOVA results for each subscale of the QOLI (N = 83).

Scale	Group					
	PTSD	ASD	Subclinical	One way ANOVA		
	M (SD)	*M (SD)*	*M (SD)*	*F (2,80)*	*p*	Post hoc
Health	-1.76 (3.03)	3.17 (3.55)	3.41 (3.21)	24.58	<.0001*	P<A,S
Self esteem	0.52 (3.04)	4.22 (2.10)	4.26 (2.72)	16.21	<.0001*	P<A,S
Goals & values	1.55 (2.65)	3.26 (1.57)	3.00 (1.69)	5.70	.005	P<A,S
Money	0.14 (2.43)	1.43 (1.56)	2.07 (1.81)	8.00	.0007*	P<A,S
Work	-0.07 (2.73)	2.22 (1.99)	3.00 (1.25)	17.18	<.0001*	P<A,S
Play	-0.72 (3.14)	3.26 (1.91)	3.90 (1.47)	36.01	<.0001*	P<A,S
Learning	1.24 (2.60)	3.65 (2.10)	4.14 (1.96)	14.40	<.0001*	P<A,S
Creativity	0.97 (2.70)	1.61 (1.92)	1.34 (1.93)	0.95	.39	n.a.
Helping	1.59 (2.11)	3.43 (2.74)	3.45 (2.56)	5.66	.005	P<A,S
Love	-0.10 (4.75)	3.78 (3.20)	3.14 (4.02)	8.88	.0007*	P<A,S
Friends	2.59 (2.51)	5.04 (1.64)	4.93 (2.09)	13.35	<.0001*	P<A,S
Children	2.28 (2.91)	3.78 (2.41)	3.21 (2.93)	2.23	.12	n.a.
Relatives	1.17 (2.61)	4.35 (2.17)	4.07 (2.64)	13.18	<.0001*	P<A,S
Home	2.38 (2.33)	4.00 (2.69)	3.86 (2.15)	4.13	.02	P<A,S
Neighbourhood	1.10 (2.28)	1.22 (2.11)	0.66 (1.39)	0.65	.53	n.a.
Community	1.45 (3.22)	3.43 (2.10)	3.03 (2.78)	4.36	.02	P<A,S

* Significant after Bonferroni correction (.05/16 = .003).

Participants were asked during the structured interview (PACI; Holmes, 1997b) if they felt that their social or working life had been affected by the MVA (No, Yes +, Yes -). A significantly greater number of the PTSD group reported that social, χ^2 (4, N = 83) = 22.01, p > .0001, and working life, χ^2 (4, N = 83) = 27.05, p < .0001, had been negatively affected by the MVA than the ASD and subclinical groups. Figures 16 and 17 display the percentage of each group reporting positive, negative or no change in these areas of functioning following the MVA.

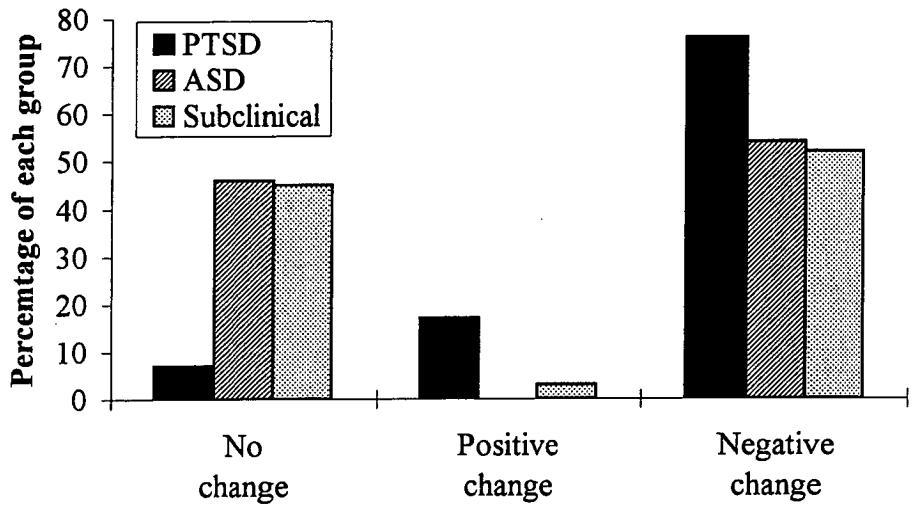

Figure 16.

Perceived posttraumatic changes in social life (% of each group) (N = 83).

Figure 16 shows that over 50% of the participants in each group reported that the MVA had negatively affected their social life. A minority of the PTSD (16%) and subclinical (3%) groups reported that the MVA had a positive effect on their social life, and the remaining participants reported that the MVA had no effect on their social life.

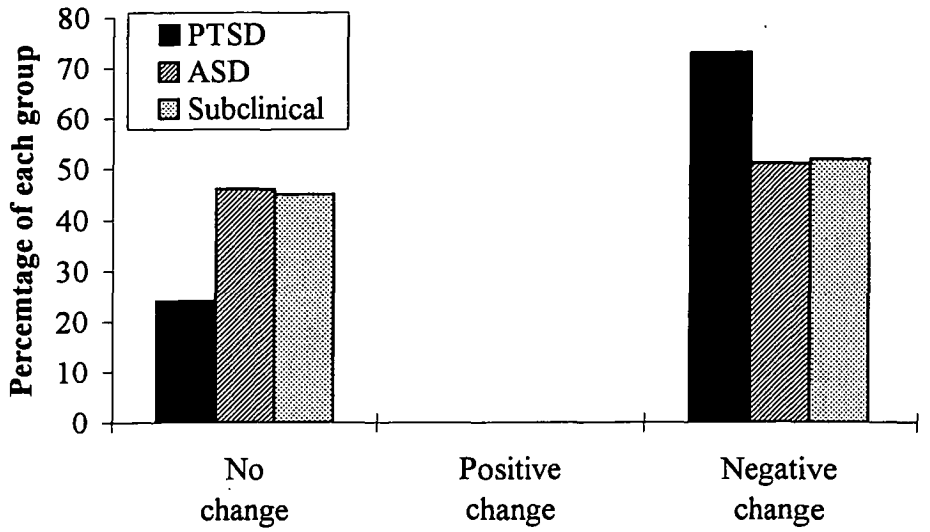

Figure 17.

Perceived posttraumatic changes in working life (% of each group) ($N = 83$).

Figure 17 shows that over 50% of the participants in each group reported that the MVA had negatively affected their working life. No participants reported that the MVA had a positive effect on their working life. The remaining participants reported that the MVA had no effect on their working life.

8.4 Discussion

8.4.1 DSM-IV criteria

The findings reinforced the classification of the three distinct diagnostic groups based on the DSM-IV criteria (APA, 1994). The analysis of the specific DSM-IV posttraumatic criteria demonstrated that the PTSD group was relatively low in reported dissociative phenomena, and may have implications for suggesting

a mechanism by which different aetiological pathways are determined. It was found that, in addition to the symptoms associated with diagnoses of PTSD and ASD, panic attacks and Major Depressive Episodes were reportedly experienced by individuals in each group, all of whom had no reported history of these symptoms prior to the trauma. This finding is consistent with previous comorbidity studies (e.g., Blanchard, Buckley, et al., 1998; Blanchard, Hickling, Taylor, & Loos, 1995; Bremner, 1999a; Bryant, 1998; Shalev, 2000). The subclinical group was generally not asymptomatic, reporting symptoms meeting some of the criteria for PTSD and ASD, and a minority fulfilling the criteria for posttraumatic panic attacks and Major Depressive Episodes.

It was considered interesting that the resolution of posttrauma symptoms in the ASD group was associated with the continued experience of little other symptomatology, and this group was less symptomatic in the longer term than the subclinical group. It was also noted that the ASD group reported significantly greater endorsement of two of the PTSD avoidance and numbing criteria than the other groups, inability to recall aspects of the trauma and emotional detachment, that have been proposed to belong in a dissociative rather than avoidance and numbing symptom cluster (e.g., van der Kolk, McFarlane, et al., 1996; van der Kolk, Pelcovitz, et al., 1996). These results provide further evidence that ASD may be considered a separate diagnostic entity from PTSD, associated with resilience and adjustment (e.g., Koopman et al., 1995), and that it is not necessarily a predictor of PTSD as suggested in some literature (e.g., Bryant et al., 1998; Classen et al., 1998; Harvey & Bryant, 1998). It is noted that many individuals in other studies have been diagnosed with ASD and PTSD (e.g., Birmes et al., 2001; Holeva et al., 2002; Winston et al., 2002), however this

progression was not evident in this sample, as discussed in chapter six. The question remains as to whether the high numbers of individuals in other studies progressing from ASD to PTSD would be better described as having a preliminary form of PTSD, as opposed to a diagnosis of ASD.

8.4.2 Current psychopathology

Given that the ASD group diagnoses were retrospective due to experimental necessity, measures such as current psychopathology were considered relevant in assessing post ASD adjustment, rather than the symptoms experienced during the course of the disorder. As hypothesized, current psychopathology measures demonstrated that the PTSD group reported significantly greater posttraumatic anxiety and depressive symptoms than the ASD and subclinical groups, which is not surprising considering that all PTSD diagnoses were current, not retrospective. The total IES-R, BAI and BDI scores of the PTSD group were reflective of clinically significant levels of psychopathology at the time of testing. The criterion for clinical caseness was fulfilled by the PTSD group in response to the SCL-90-R scales Somatization, Obsessive-compulsive, Depression, Anxiety and Interpersonal sensitivity. The ASD and subclinical group scores across the SCL-90-R scales were below the clinical threshold, relecting that these groups were not reporting distress caused by clinically significant psychopathology at the time of testing. Again, the apparent completion of recovery of the ASD group was noted with interest, and the results were found to be supportive of the second hypothesis.

8.4.3 Psychometric profiles of psychopathology and personality

Responses to the PAI and TSI reflected valid assessments. Clinically significant elevations in the moderate range were found for the PTSD group on the Somatic Complaints, Anxiety, Anxiety-Related Disorders, Depression and Schizophrenia scales of the PAI in support of the first hypothesis. The PTSD group also scored significantly more highly than the other groups on the following TSI clinical scales from the dysphoric mood and posttraumatic stress categorizations of the TSI: Anxious Arousal, Intrusive Experiences, Defensive Avoidance and Dissociation, also in support of the first hypothesis. The ASD and subclinical group scores were not significantly different on these scales.

The PTSD group scored significantly more highly than the subclinical group on the TSI dysphoric mood category dimensions, the Depression and Anger/Irritability scales. The PTSD group scores on the Anxious Arousal, Intrusive Experiences and Defensive Avoidance subscales were clinically significant. The similarity between the TSI Tension Reduction Behaviour scale scores of the groups may reflect the tendency of the PTSD group to not reduce their high levels of anxiety by engaging in tension reducing behaviours.

There was a significant main effect for the Negative Relationships subscale of the Borderline Features scale, and analyses showed that the PTSD group scored significantly higher than the ASD and subclinical groups on this subscale, and in the moderate clinical range. In addition, there was a significant main effect for the Egocentricity subscale of the Antisocial Features scale, with analyses showing that the ASD group scored significantly higher than the subclinical and PTSD groups on this subscale prior to Bonferroni correction. However, the scores were all within the average range. The Warmth scale of the PAI assessed the extent to

which a person may be considered empathic and engaging in interpersonal relationships. Analyses showed that the ASD group scored significantly higher than the PTSD group on this scale, however, all group scores were also within the normal range. These results suggest that normal deviations in personality traits may differentiate the ASD group from the other groups, and potentially may play a mediating role in posttraumatic adjustment. It may be speculated that the personality concepts of Egocentricity and Warmth, as defined in the development of the PAI, are associated with posttraumatic adjustment. The PTSD profile demonstrated significantly greater self-report of negative relationships as measured by the borderline characteristics subscale, and non-psychotic symptoms of schizophrenia, specifically relating to social detachment and concentration difficulties. It was not possible to determine from the data if personality trait differences preceded, or resulted from, trauma exposure. Although the relationship between social support and psychiatric disorder is complex, there is general agreement that social support assists and reflects more adaptive adjustment (e.g., Henderson, Byrne, & Duncan-Jones, 1981).

8.4.4 Biopsychosocial outcome measures

There were no significant between group differences in reported changes in driving behaviour and attitude to traveling by road. The majority of the participants in each of the three groups reported that the MVA had negatively affected the way they felt about driving and traveling by road, and negatively affected their behaviour, consistent with the findings of Hickling, Blanchard, Buckley, et al., (1999). Less than one quarter of each group reported positive improvement in attitudes and behaviours related to driving and traveling by road.

The findings in relation to predicted psychophysiological response to driving related situations indicated group specific modes of response. The self-reported predictions of modes of psychophysiological response to potentially anxiety provoking driving-related situations corroborated the group specific modes of actual psychophysiological response in the previous study. It cannot be distinguished from the data if the group specific modes of response preceded the traumatic experience.

There were no significant group differences found in reported posttraumatic changes in potentially addictive behaviors. This finding indicates that substance use did not influence differential posttraumatic psychological pathways for the MVA survivors, as also noted by Blanchard and Hickling (1997). There were significant between group differences in posttraumatic perceived quality of life. The ASD group profile reflected positive posttraumatic adjustment, in support of the second hypothesis. The PTSD group perceived overall quality of life to be significantly less than the other groups, consistent with the findings of Hickling, Blanchard, Mundy, et al., (1999), and supporting the third hypothesis.

Specifically, Health, Self-esteem, Money, Work, Play, Learning, Love, Friends and Relatives were rated by the PTSD group to be significantly less satisfying than the other groups. A significantly higher number of the PTSD group reported that social and working life had been negatively affected by the MVA than the ASD and subclinical groups.

As it is a requirement, by definition, that PTSD is associated with clinically impairment of social and occupational functioning, these results were not surprising, and have been found in previous studies (e.g., Cordova et al., 1995; Hickling, Blanchard, Mundy, et al., 1999; Zatzick et al., 1997). However, it is

interesting to note the extent to which the PTSD group reported pervasive reduction in quality of life, and to contemplate the potential long term impact of enduring reduced quality of life. Although it cannot be ascertained from the data, it is also worth consideration that objective differences in these quality of life variables, for example, poorer health, financial disadvantage, unemployment, or lack of social support may have been more prevalent in the PTSD group than the other groups, and these variables may have mediated posttraumatic outcomes. As suggested in the literature (e.g., Huyse et al., 2001), the identification of areas of biopsychosocial dissatisfaction may facilitate appropriate intervention and treatment targets. In this case, the PTSD group may be considered to potentially benefit from psychological therapy targeting variables such as activity level, support networks, and self-concept.

8.4.5 Summary and conclusions

The findings supported the classification of three distinct diagnostic groupings based on DSM-IV criteria (APA, 1994). The findings regarding psychopathology associated with posttraumatic responses supported the existing literature regarding PTSD and DSM-IV Axis I comorbidity (e.g., O'Brien, 1998; Scott & Stradling, 1992), and also reflected a degree of overlap in diagnostic criteria for these disorders. These findings have demonstrated that posttraumatic responses to MVA trauma vary from non-psychopathological adjustment to the extreme of the development of PTSD and associated psychopathology, in support of previous literature (e.g., Blanchard & Hickling, 1997; Hickling, Blanchard, Mundy, et al., 1999). These findings reinforce the status of MVA trauma as a significant stressor which can result in the development of psychopathology in

individuals free from pretrauma psychopathology. The results support the hypothesis that MVA trauma can result in the development of the full range of posttraumatic symptoms and associated psychopathology, and the specific hypotheses proposed at the commencement of this study.

These results supported previous profiles using psychometric measures other than the PAI (e.g., Blanchard, Hickling, et al., 1998; Deering et al., 1996). The profiles of both the ASD and subclinical groups were characterized by all scales measuring within the average range, reflecting emotional stability and adaptive functioning, and indicating that the initial diagnosis of ASD was associated with longer term adaptive functioning. The exploratory findings in relation to greater interpersonal warmth and egocentricity of the ASD group in comparison to the PTSD group, indicated that these personality characteristics may influence avoidance of the development of PTSD. Further investigation of these variables may be warranted.

It was proposed that the personality profiles of the subset of individuals with a retrospective diagnosis of ASD without progression to PTSD reflect adaptive recovery in the longer term, challenging the status of this short term response as 'disordered'. Although the symptoms of ASD are distressing in the short term, as defined by the criteria, it may be speculated that the early symptoms occurring within the first month after trauma exposure are reflective of productive processes, such as cognitive processing of traumatic memories, that assist posttraumatic adjustment in the longer term.

All groups experienced the psychological impact of traumatic MVAs, however, it was evident that the impact of MVA trauma was pervasive and enduring for the PTSD group, as measured by a broad range of psychopathological

and biopsychosocial outcomes. Despite the suggestion that ASD is a precursor to PTSD, this study has provided further evidence of a subset of individuals who were diagnosed with ASD, yet demonstrated a longitudinally resilient and adaptive posttraumatic recovery without progression to PTSD, as suggested by Bryant and Harvey (2000a). It was proposed that this group provides a focus for further examination of the transition from psychopathological distress to resilience, and challenges the classification of the posttraumatic response of these individuals as 'disordered' according to the medical model. This study has provided evidence of the alternative pathways of adaptation and resilience versus psychopathological and biopsychosocial negative outcomes, as proposed in the literature (e.g., Beardslee, 1989; Lyons, 1991; McFarlane & Yehuda, 1996; van der Kolk & McFarlane, 1996; Valent, 1999). The findings were consistent with the integrated model of the aetiology of posttraumatic stress disorders, as they demonstrate cognitive, affective, behavioural and physical differences measured by posttraumatic variables that are associated with the differential development of posttraumatic stress disorders.

This study has been previously presented in part, and referenced contributions by the author of this thesis are underlined:

Holmes, G.E., Williams, C.L., & Haines, J. (1998g, September). *Posttraumatic psychopathology following motor vehicle accidents.* Paper presented at the World Federation for Mental Health Asia Pacific Regional Conference, Hobart, Australia.

Holmes, G.E., Williams, C.L., & Haines, J. (1998h). Posttraumatic psychopathology following motor vehicle accidents. *Open Mind: Journal of the Tasmanian Association for Mental Health, 21,* 16.

Holmes, G.E., Williams, C.L., & Haines, J. (1998i). Posttraumatic psychopathology following motor vehicle accidents. *Mental Health in Australia: Journal of the Australian National Association for Mental Health, 47,* 32-38. (Appendix E-3)

Holmes, G.E., Williams, C.L., & Haines, J. (2001b). Motor vehicle accident trauma exposure: Personality profiles associated with posttraumatic diagnoses. *Anxiety, Stress & Coping, 14,* 301-313. (Appendix E-4)

CHAPTER NINE

SUMMARY AND CONCLUSIONS

9.1 Summary of results

This investigation had two main objectives. The first was to produce multi-variable psychological profiles associated with the development of PTSD, ASD and subclinical posttraumatic responses. This process aimed to increase understanding of the development of diagnostically distinct psychological responses to trauma, and to investigate variables associated with posttraumatic adjustment following ASD. This objective was achieved. The second objective was to specifically focus on the psychological impact of MVA trauma. MVA trauma was found to be a frequently occurring trauma type in the Tasmanian community, that may result in decreased quality of life and compromised mental health. MVA trauma was found to result in PTSD, ASD and subclinical responses, with associated comorbidity. It was also noted that exposure to a traumatic MVA did not always result in the development of posttraumatic symptoms, and posttraumatic psychological benefits such as personal growth were acknowledged. Thus, the second objective was also achieved.

This series of studies showed PTSD and ASD to be distinct entities warranting separate psychiatric classifications. The results demonstrated that ASD without progression to PTSD may be considered a diagnosis predictive of longer term psychological adaptation, following an initial period of psychiatric symptomatology. It was speculated that the ASD group, despite initial symptoms, were influenced by stress moderating variables that protected them from long term negative outcomes. The investigation presented evidence that the psychological impact of MVA trauma can result in a range of responses from adaptive recovery to chronic psychiatric illness.

A broad range of theoretical models of the aetiology of posttraumatic stress disorders were presented to exemplify the complexity of the differential development of posttraumatic responses, and their longitudinal course. The literature indicated that all of the aetiological models had strengths, and have contributed to the understanding and recognition of mediating variables in the differential development of posttraumatic responses. However, the majority of the aetiological theories did not consider ASD. If ASD is a separate entity, as suggested by the results of the present investigation, it would be expected to have a different aetiology than PTSD. The literature review identified a need for efforts to be directed towards determining the aetiology of ASD.

It was concluded that no single theory explained the complexity of the aetiology of posttraumatic stress disorders. An integrated model to represent the influence of multiple internal and external variables in the aetiology of differential posttraumatic responses was proposed. Consistent with the model, cognitive, affective, behavioural, and physical variables were found to differentiate the distinct DSM-IV (APA, 1994) classifications of posttraumatic stress disorders.

Prior to investigating the variables associated with PTSD, ASD and subclinical responses to MVA trauma, it was established in the first study that the majority of the sample reported exposure to an MVA. Many of those who had been exposed to an MVA reported the experience to be psychologically traumatic. Exposure rates to traumatic MVAs conflicted with the suggestion that only a minority of MVAs at the most severe end of the objective spectrum could be considered traumatic events, and it was speculated that this may be the result of different ideas about what experiences meet the criteria for a traumatic event. Fear of death and serious injury experienced in response to the MVAs were found to

exceed objective measures of MVA-related injuries and deaths, supporting the importance of subjective perceptions rather than objective statistics in the definition of a traumatic event. The full spectrum of posttraumatic symptoms was reported within the sample. The males in the sample were found to have comparable lifetime exposure rates to MVAs with other studies, however, the females in the sample were found to have almost three times greater risk of exposure to a traumatic MVA than American females. This finding was interpreted in terms of potentially mediating variables, such as the result relating more to being a passenger in the MVA than being female. The results indicated that traumatic MVA exposure occurred with sufficient frequency in this population to make a series of studies of diagnostically distinct posttraumatic responses to MVA trauma a viable option.

Given that it was indicated in the findings of the first study that females reported greater prevalence of exposure to MVAs and posttraumatic symptoms, a result supported in the literature, it was not surprising that there were more females than males in the diagnostic groups used for the subsequent three studies. On the basis of the proposed integrated model of the aetiology of posttraumatic stress disorders, it was decided that multimodal assessment of posttraumatic responses was necessary to comprehensively examine a selection of the wide range of variables reported to influence posttraumatic response. This aim was achieved in studies two to four, which provided an examination of multiple cognitive, affective, behavioural, and physical variables, and their relationships to the development of distinct posttraumatic psychological responses.

The results of study two indicated that the aetiologies of PTSD and ASD were associated with different coping style profiles, but not different belief systems. No significant differences were found in the coping and belief profiles of the ASD

and subclinical groups to explain the aetiology of these distinct posttraumatic pathways. The distinction between the coping style profiles of the PTSD and ASD groups provided information about the role of adaptive coping strategies in recovery from posttraumatic symptoms associated with the ASD diagnosis. However, as highlighted by the integrated aetiological theory of posttraumatic stress disorders, posttraumatic adjustment pathways are based on multiple variables. Study three shifted the focus from coping and beliefs to recall of traumatic memories, in the multimodal investigation of the development of diagnostically distinct posttraumatic responses to MVA trauma.

Study three identified psychophysiological and psychological patterns that may be influential in the differential development of PTSD, ASD and subclinical posttraumatic responses. Many diagnosis, script, and stage specific response patterns were found, including situation specific reactivity of all groups, elevated psychophysiological arousal in response to recalling the MVA, integrated psychophysiological and psychological evidence of dissociation occurring during the course of the recall of the event, and the most negative psychological responses occurring during recall of the aftermath at the accident scene. These findings were evaluated as potentially useful in vulnerability prediction and early intervention. The four stage guided imagery methodology of integrated psychophysiological and psychological assessment was proposed as a clinical tool that may have utility in the diagnosis, assessment and treatment of posttraumatic responses. The methodology is an advancement of previous imagery tools, in that it allows patterns of responses to specific aspects of recall of an experience to be measured and assessed.

As proposed in the integrated aetiological model, the investigation of associations between posttraumatic variables and diagnostic outcomes was the final three-phase component of the multimodal assessment. This investigation was carried out in the final study. The findings of study four demonstrated that posttraumatic responses to MVA trauma vary from non-psychopathological adjustment to the extreme of the development of PTSD and associated psychopathology. The findings reinforced the status of MVA trauma as a significant stressor which can result in the development of psychopathology. The profiles of both the ASD and subclinical groups were characterized by emotional stability and adaptive functioning, and indicated that the initial diagnosis of ASD was associated with longer term adaptive functioning. It was proposed that the posttraumatic personality profiles of the subset of individuals with a retrospective diagnosis of ASD without progression to PTSD reflected adaptive recovery in the longer term, challenging the status of this short term response as 'disordered'. The exploratory findings in relation to greater interpersonal warmth and egocentricity of the ASD group in comparison to the PTSD group, indicated that these personality characteristics may have influenced avoidance of the development of PTSD.

It was apparent from the results that all groups experienced the psychological impact of traumatic MVAs. However, it was evident that the impact of MVA trauma was pervasive and enduring for the PTSD group, as measured by a broad range of biopsychosocial outcomes. Despite the suggestion that ASD is a precursor to PTSD, the present investigation provided evidence that the ASD group experienced a longitudinally resilient and adaptive posttraumatic recovery without progression to PTSD at the time of the assessment.

It is proposed that this group provides a focus through which the transition from psychopathological adjustment to resilience may be further examined, and challenges the classification of the posttraumatic response of these individuals as 'disordered' according to the medical model. The results reflect that the ASD group may benefit from support during the first month following the event as they are experiencing distress during that time. Due to this distress, it may be considered that a psychiatric diagnosis is warranted, however, it is proposed that diagnoses within the first month should be considerate of adaptive versus maladaptive processes underlying the distress. It is speculated that initial distress that is caused by processes such as cognitive processing of traumatic memories and confronting subjective experiences such as guilt and fear represent preparation for adaptation.

9.2 Critical analysis of the investigation

A primary strength of this research was the heterogeneous sample of MVA survivors. Rather than using a sample of treatment-seeking survivors, the community sample provided a broader range of participants that were not necessarily seeking psychological treatment. However, a limitation of using a sample of volunteers was the potential bias of such a sample who were willing to come forward and speak about their trauma and its psychological impact. Strengths of the sample used were the demographic similarities between the groups and stringent exclusion participant selection criteria, reducing problems associated with factors such as sex and age differences in response.

A longitudinal design following participants from MVA throughout the following months or years would have been ideal, however, a cross-sectional study was more viable given the available resources. In terms of measures used, as previously discussed, the inclusion of self-report measures may be considered to have both strengths and limitations. As mood and situational factors can bias self-report (e.g., Eich, 1995; Wyshak, 1994), the degree to which these elements affected the data presented remains unknown. However, as previously discussed, the utilization of the selected tools has been endorsed as appropriate (e.g., Wilson & Keane, 1997).

9.3 Implications for the assessment, diagnosis and treatment of posttraumatic responses

This investigation identified that MVAs are a frequently occurring trauma type in Tasmania, yet participants reported a low participation rate in posttraumatic psychological counselling. Although it is acknowledged that formal psychological approaches are not necessarily the best approach for all individuals exposed to trauma (e.g., Scott & Stradling, 1992), this finding highlighted the level of need for psychological support within the community following MVAs. On the basis of this research, an information booklet for survivors of MVA trauma was designed, and sponsored by the Motor Accidents Insurance Board for statewide distribution within Tasmania, in order to provide information regarding professional and family support strategies. The booklet is displayed in Appendix F-1.

Although the present investigation exclusively examined MVA trauma, it is proposed that the selection of tools, including the newly adapted four stage guided

imagery methodology, may be utilized to examine other trauma types and populations. Previous findings have suggested that exposure-based and biofeedback therapies using imagery techniques can be an effective treatment of posttraumatic symptoms (e.g., Blanchard & Abel, 1976; Ehlers, 1999; Ehlers et al., 1998; Foa, Keane, & Friedman, 2000; Forbes et al., 2001; Harvey, 1999; Keane, 1995; Keane, 1997; Keane, Street, & Orcutt, 2000; Nathan & Gorman, 1998; Sherman, 1998). It is proposed that further research may identify that a four stage guided imagery methodology may also have considerable treatment utility as a form of therapeutic exposure. Anecdotally, the participants in this research reported increased self-efficacy after coping with the process of providing the description of their experiences and imaging the scenes. Such experiences in a treatment program may provide mastery experiences and result in the reduction of psychiatric symptoms, as proposed by Bandura, Reese, and Adams (1982).

9.4 Directions for future research

Despite the fact that the DSM-IV (APA, 1994) diagnostic categories of PTSD and ASD are not trauma-specific, it may be prudent to explore the findings relating to these diagnoses using populations exposed to other trauma types. In addition, it would be an interesting extension of this series of studies to explore age and sex differences within the diagnostic groups. This was not possible in the series of diagnosis-specific studies due to the nature of the sample.

It is proposed that longitudinal examination of the identified group differences found, in terms of time elapsed posttrauma, may be a direction for future research to explore the stability of these differences over time. In addition,

the nature of the three diagnostic groups used may be considered heterogeneous in diagnostic terms, such as the inclusion of acute and chronic PTSD in one group, and varying symptoms profiles within the subclinical group. Ideally, a symptom-free group and an ASD with progression to PTSD group would have also been included in this investigation for comparative purposes, however, these responses to MVA trauma were not found in the sample obtained. It is proposed that future research may include comparisons of symptom-free individuals following trauma exposure, acute versus chronic versus delayed PTSD, and the examination of the subset of individuals who develop ASD which progresses to PTSD.

In terms of tools for further research, as acknowledged in chapter four, it would have been desirable to have access to neuroimaging technology as part of the multimodal assessment battery. It is proposed that a combination of the four stage guided imagery methodology and neuroimaging techniques may provide a more advanced method in the future of examining differences in cerebral activity occurring during recall of traumatic and nontraumatic memories. This combined assessment methodology may be a promising area for future research, and further increase understanding of the differential development of diagnostically distinct posttraumatic responses.

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APPENDICES

APPENDIX A

ADDITIONAL INFORMATION: CHAPTER TWO

Appendix A-1: Diagnostic criteria for PTSD (APA, 1994).

A. The person has been exposed to a traumatic event in which both of the following were present:

(1) the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others.

(2) the person's response involved intense, fear, helplessness, or horror.

Note: in children, this may be expressed instead by disorganised or agitated behaviour.

B. The traumatic event is persistently reexperienced in one (or more) of the following ways:

(1) recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions.

Note: In young children, repetitive play may occur in which themes or aspects of the trauma are expressed.

(2) recurrent distressing dreams of the event.

Note: In children, there may be frightening dreams without recognisable content.

(3) acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated).

Note: In young children, trauma-specific re-enactment may occur.

(4) intense psychological distress at exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event.

(5) physiological reactivity on exposure to internal or external cues that symbolise or resemble an aspect of the traumatic event.

C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:

(1) efforts to avoid thoughts, feelings, or conversations associated with the trauma.

(2) efforts to avoid activities, places, or people that arouse recollections of the trauma.

(3) inability to recall an important aspect of the trauma.

(4) markedly diminished interest or participation in significant activities.

(5) feeling of detachment or estrangement from others.

(6) restricted range of affect (e.g., unable to have loving feelings).

(7) sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span).

D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following:

(1) difficulty falling or staying asleep.

(2) irritability or outbursts of anger.

(3) difficulty concentrating.

(4) hypervigilance.

(5) exaggerated startle response.

E. Duration of the disturbance (symptoms in Criteria B, C, and D) is more than 1 month.

F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specify if:

Acute: if duration of symptoms is less than 3 months.

Chronic: if duration of symptoms is 3 months or more.

With Delayed Onset: if onset of symptoms is at least 6 months after the stressor.

Appendix A-2: Diagnostic criteria for ASD (APA, 1994).

A. The person has been exposed to a traumatic event in which both of the following were present:

(1) the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others.

(2) the person's response involved intense, fear, helplessness, or horror.

B. Either while experiencing or after experiencing the distressing event, the individual has three (or more) of the following dissociative symptoms:

(1) a subjective sense of numbing, detachment, or absence of emotional responsiveness.

(2) a reduction in awareness of his or her surroundings (e.g., "being in a daze").

(3) derealization.

(4) depersonalisation.

(5) dissociative amnesia (i.e., inability to recall an important aspect of the trauma).

C. The traumatic event is persistently reexperienced in at least one of the following ways: recurrent images, thoughts, dreams, illusions, flashback episodes, or a sense of reliving the experience; or distress on exposure to reminders of the traumatic event.

D. Marked avoidance of stimuli that arouse recollections of the trauma (e.g., thoughts, feelings, conversations, activities, places, people).

E. Marked symptoms of anxiety or increased arousal (e.g., difficulty sleeping, irritability, poor concentration, hypervigilance, exaggerated startle response, motor restlessness).

F. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning or impairs the individual's ability to pursue some necessary task, such as obtaining necessary assistance or mobilising personal resources by telling family members about the traumatic experience.

G. The disturbance lasts for a minimum of 2 days and a maximum of 4 weeks and occurs within 4 weeks of the traumatic event.

H. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition, is not better accounted for by Brief Psychotic Disorder, and is not merely an exacerbation of a pre-existing Axis I or Axis II disorder.

APPENDIX B

ADDITIONAL INFORMATION: CHAPTER FIVE

Appendix B-1:

The Motor Vehicle Accident Questionnaire [MVAQ] (Holmes, 1997a)

Motor Vehicle Accident Research

Conducted by: Georgina Holmes (Doctoral student in Clinical Psychology) under the supervision of Dr Chris Williams.

We are collecting data regarding the prevalence and nature of motor vehicle accidents involving the student population. We would greatly appreciate your time and honesty in completing the following questions.

1. Age: _____
2. Sex: _____
3. Have you ever been involved in a motor vehicle accident? Yes/No
   If so, please answer the following questions.

(If you have been involved in more than one motor vehicle accident, please answer the following questions in relation to the accident you felt was the worst psychological experience for you.)

4. Did the accident cause you to feel intense negative emotions such as fear, helplessness or horror? Yes/No

5. How old were you when the accident occurred?

6. When did the accident occur (month & year if possible)?

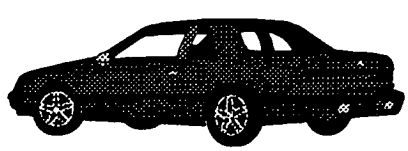
7. What was your role in the accident (driver/passenger/pedestrian/other)?

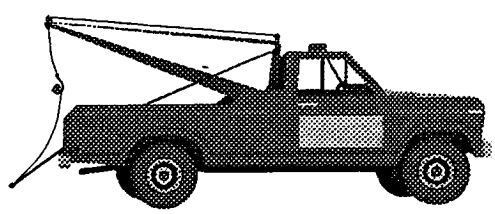
8. How many vehicles were involved in the accident?

9. How many people were injured in the accident?

10. How many people were killed in the accident ?

11. Were you injured in the accident? *If so, please describe in brief the injuries you received.*





12. At the time of the accident, did you fear that you were going to die? Yes/No

13. Did you fear that someone else involved was going to die? Yes/No

14. Did you fear that you were going to be seriously injured? Yes/No

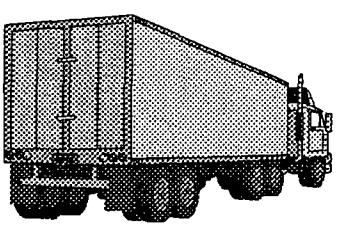
15. Did you fear that someone else was going to be seriously injured? Yes/No

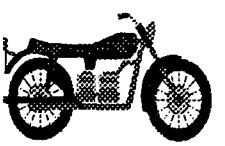
16. In response to the accident did you experience any of the following *(please tick)*

- ☐ a) Distressing dreams
- ☐ b) Distressing flashbacks
- ☐ c) Distressing reminders of the accident
- ☐ d) Trying to avoid reminders of the accident
- ☐ e) Trying to avoid thinking or talking about the accident
- ☐ f) Feeling flat and unable to react emotionally
- ☐ g) Sleeping and/or concentration difficulties
- ☐ h) Increased irritability or anger
- ☐ i) Feeling jumpy
- ☐ j) Feeling in a daze
- ☐ k) Feeling numb
- ☐ l) Feeling unreal or detached from your surroundings

Would you be interested in participating further in our research? If so, please write your first name and contact phone number below.

Thank you for your time and assistance.





Appendix B-2:

MVAQ response summary of the nontraumatic MVA subgroup (n = 106)

MVAQ item	n (%)
Fear of death and injury	
At the time of the accident, did you fear that you were going to die?	0 (0)
Did you fear that someone else involved was going to die?	0 (0)
Did you fear that you were going to be seriously injured?	1 (1)
Did you fear that someone else was going to be seriously injured?	1 (1)
Reexperiencing symptoms	
1. Distressing dreams	10 (9)
2. Distressing flashbacks	8 (8)
3. Distressing reminders of the accident	10 (9)
Avoidance and numbing symptoms	
4. Trying to avoid reminders	8 (8)
5. Trying to avoid thinking/talking about the MVA	10 (9)
6. Feeling flat and unable to react emotionally	12 (11)
Hyperarousal symptoms	
7. Sleeping and/or concentration difficulties	10 (9)
8. Increased irritability or anger	8 (8)
9. Feeling jumpy	15 (14)
Dissociative symptoms	
10. Feeling in a daze	22 (21)
11. Feeling numb	16 (15)
12. Feeling unreal or detached	11 (10)

APPENDIX C

ADDITIONAL INFORMATION: CHAPTER SIX

Appendix C-1 : Participant information sheet and consent form

Information Sheet

Investigation of psychological responses to motor vehicle accidents.

The above project is currently being conducted by Dr Chris Williams and Miss Georgina Holmes of the Department of Psychology at the University of Tasmania. The purpose of the project is to learn more about psychological responses to motor vehicle accidents. In particular, we are interested in examining how motor vehicle accidents affect the lifestyle, thoughts and actions of the people involved. The aim is to develop better ways of helping people recover psychologically after motor vehicle accidents.

We are interested in making comparisons between adults (18 years +) who develop anxiety or mood disorders after a motor vehicle accident, and those who display few, if any, adverse reactions. These comparisons will draw on the nature and consequences of the accident, thoughts about the accident, actions performed in response to the accident, psychological symptoms of distress, and bodily responses to imaging the accident. If you agree to participate, the nature of your accident, and your response to it, will be discussed with you. Confidentiality between participants and the investigators will be maintained by the use of participant numbers on data files. The investigators are Clinical Psychologists, thus professional support will be provided for all participants throughout the investigation.

Should you choose to be involved in this project you will be asked to participate in a maximum of three sessions (each one hour in duration) :

1. Interview 1: You will be interviewed about the accident you were involved in, and how you have responded to it. You will be asked to take home some questionnaires to complete at your leisure.
2. Interview 2: You will be interviewed regarding three events you have experienced (a neutral event such as making coffee, an arousing event such as exercise, and the accident). This interview will be recorded on audio cassette so that the investigator can design personalized guided imagery scripts.
3. Laboratory session: Your bodily responses (such as heart rate and muscle tension) will be measured while you sit and picture the events that you described using the guided imagery scripts. Electrodes will be applied to facilitate measurements.

Participation is voluntary, and participants who decide to take part in the study may withdraw at anytime by stating a wish to do so. The project has received ethical approval from the University Ethics Committee (Human Experimentation). However, should participants have ethical concerns about the project that they wish not to discuss with the investigators, they may contact the Chair or Executive Officer of the University Ethics Committee (Human Experimentation). Results of the investigation will be available on request at the conclusion of the project.

If you would like to participate in this research, please contact:
Georgina Holmes
☎ (03) 62 262261.

Statement of informed consent

Correlates of diagnostically distinct psychological responses to motor vehicle accidents.

I _____ consent to participate in the study being conducted by Dr Chris Williams and Miss Georgina Holmes. I understand that the study is being conducted in an attempt to understand the effects of exposure to a serious motor vehicle accident. I understand that I have been asked to participate in this study to compare my responses with other individuals who have been exposed to similar trauma. I understand that I will be expected to answer questions about my experience of a motor vehicle accident and how I have reacted to this experience. I also understand that measurements of my heart rate, breathing and blood pressure will be taken while I am asked to imagine a series of situations that I have selected. I understand that all research data will be treated as confidential. I understand that I may withdraw from the study at any time by stating a wish to do so. I also understand that if I have any concerns about the study I may discuss these concerns with the investigators Dr. Chris Williams or Georgina Holmes on (03) 62 262245. I have read and understood the 'Information Sheet' for this study.

I have read the information above and any questions I have asked have been answered to my satisfaction. I agree to participate in this investigation and understand that I may withdraw at any time. I agree that research data gathered for the study may be published provided that I cannot be identified as a participant.

Name of participant: _____
Signature of participant: _____
Date: _____

I have explained this project and the implications of participation in it to this volunteer and I believe that the consent is informed and that he/she understands the implications of participation.

Name of Investigator: _____
Signature of Investigator: _____
Date: _____

Post-Accident Clinical Interview

Demographic Details

1. Participant code		6. Level of education	
2. Gender	Male (1) / Female (2)	7. Postcode	
3. Current age		8. Date of MVA	
4. Age at MVA		9. MVA - Interview time	
5. Marital status	Single (1) Married or De Facto (2) Separated/Divorced/Widow (3)	10. MVA - Lab time	

Accident Details

ITEMS	QUALITATIVE DATA	QUANTITATIVE DATA
11. What was your role in the accident?		Driver (1)/ Passenger (2)/Other(3)
12. How many vehicles were involved?		_____ (number)
13. Was anyone injured in the accident?		_____ (number)
14. Was anyone killed in the accident?		_____ (number)

15. Were you trapped in a vehicle?		No (1) / Yes (2)
16. How long did you remain at the accident scene?		_____ (minutes)
17. Were you unconscious at any stage after the accident?		No (1) / Yes (2)
18. Were you under the influence of alcohol or other drugs at the time of the accident?		No (1) / Yes (2)
19. Was legal action taken as a result of the accident?		No (1) / Yes (2)
20. Did you receive counselling after the accident?		No (1) / Yes (2)
21. Had you been involved in a motor vehicle accident before?		No (1)/ Yes (2)
22. Have you experienced any event that was more traumatic than the accident?		No (1) / Yes (2)

Physical Injuries and Medical History

ITEMS	QUALITATIVE DATA	QUANTITATIVE DATA
23. Were you injured in the accident?		No (1) / Yes (2)
24. Were you taken to hospital?		No (1) / Yes (2)
25. Are you currently experiencing any physical pain which was caused by the accident?		No (1) / Yes (2)
26. Do you suffer from any major illnesses?		NA
27. Do you have any physical disabilities?		NA
28. Are you currently taking any medication?		NA
29. Have you ever sustained a head injury?		NA
30. Have you ever had a mental illness?		NA

Cognitive responses to the accident

ITEMS	QUALITATIVE DATA	QUANTITATIVE DATA
31. At the time of the accident did you fear that you were going to die?		No (1) / Yes (2)
32. Did you fear that someone else involved was going to die?		No (1) / Yes (2)
33. Did you fear that you were going to be seriously injured?		No (1) / Yes (2)
34. Did you fear that someone else involved was going to be seriously injured?		No (1) / Yes (2)
35. Has the accident changed how you think or feel about yourself?		No (1) / Yes (2)
36. Did you feel guilty about any aspect of the accident?		No (1) / Yes (2)
37. Did you feel grief or loss as a result of the accident?		No (1) / Yes (2)

38. What was the worst aspect of the accident for you?		NA
39. Has the accident affected the way you feel about driving/travelling by road?		No (1) / Yes (2)
40. Has the accident changed the way you think or feel about life in general?		No (1) / Yes (2)

Behavioural Responses to the Accident

ITEMS	QUALITATIVE DATA	QUANTITATIVE DATA
41. Has the amount of alcohol you drink changed since the accident?		NA (1) / No (2) / Yes, decreased (3) / Yes, increased (4)
42. Has the amount that you smoke changed since the accident?		NA (1) / No (2) / Yes, decreased (3) / Yes, increased (4)
43. Has the amount of drugs that you take changed since the accident?		NA (1) / No (2) / Yes, decreased (3) / Yes, increased (4)
44. Has the amount that you gamble changed since the accident?		NA (1) / No (2) / Yes, decreased (3) / Yes, increased (4)

45. Has the accident affected your social life?		No (1) / Yes, positively (2) / Yes, negatively (3)
46. Has the accident affected your working life?		No (1) / Yes, positively (2) / Yes, negatively (3)
47. Has the accident affected your driving/travelling by road?		No (1) / Yes, positively (2) / Yes, negatively (3)
48. Have you returned to the scene of the accident?		No (1) / Yes (2)
49. Have you done anything which has helped you to recover from the accident?		No (1) / Yes (2)
50. Has the accident changed your lifestyle?		No (1) / Yes (2)

To be considered for each symptom item :

Has the symptom been experienced?
When was the symptom first experienced?
When was the symptom last experienced?
Was the symptom experienced before the accident?
Was the symptom due to factors other than the accident?

Posttraumatic Stress Disorder

B1 : Since the accident, have you had distressing recollections about what happened?

B2 : Have you had any distressing dreams about the accident?

B3 : Have you acted or felt as if you were reliving the accident?

B4 : When reminded of the accident, have there been times when you have become very distressed?

B5 : When reminded of the accident, have there been times when your body reacted, such as your heart thumping, sweating or tense muscles?

C1 : Have there been times when you have tried to avoid thinking, feeling, or talking about the accident?

C2 : Have there been times when you have tried to avoid activities, places or people that remind you of the accident?

C3 : Have you been unable to remember important details about the accident?

C4 : Have there been times when you have felt disinterested or stopped participating in activities you were involved in before the accident?

C5 : Have there been times since the accident when you have felt detached or estranged from the people around you?

C6 : Have there been times since the accident when you have been unable to feel emotions (such as being unable to have loving feelings)?

C7 : Have there been times since the accident when you have felt that your future is limited?

D1 : Have there been times since the accident when you have had difficulty falling or staying asleep?

D2 : Have there been times since the accident when you have been irritable or had outbursts of anger?

D3 : Have there been times since the accident when you have had difficulty concentrating?

D4 : Have there been times since the accident when you have felt highly alert to your surroundings?

D5 : Have there been times since the accident when you have been very easily startled?

Acute Stress Disorder

B1 : Have there been times since the accident when you have felt numb, detached, or unable to respond emotionally?

B2 : Have there been times since the accident when you have felt in a daze?

B3 : Have there been times since the accident when things around you did not seem real?

B4 : Have there been times since the accident when you have felt detached from yourself?

Brief Psychotic Disorder

A1 : Have there been times since the accident when people have commented that you have strange beliefs?

A2 : Have there been times since the accident when you have heard, seen, smelt, felt or tasted things that you knew were not real, such as hearing a crash or seeing blood?

A3 : Have there been times since the accident when people have commented that they cannot understand what you are saying?

A4 : Have there been times since the accident when you have felt overwhelmingly confused, or unable to move in response to your surroundings?

Panic Attacks

Have there been times since the accident when you have felt a short period of intense fear or panic?

If so, which of the following symptoms did you experience :

(1) palpitations, pounding heart, or accelerated heart rate

(2) sweating

(3) trembling or shaking

(4) sensations of shortness of breath or smothering

(5) feeling of choking

(6) chest pain or discomfort

(7) nausea or abdominal distress

(8) feeling dizzy, unsteady, lightheaded or faint

(9) derealization (feelings of unreality) or depersonalization (being detached from oneself)

(10) fear of losing control or going crazy

(11) fear of dying

(12) parathesias (numbness or tingling sensations)

(13) chills or hot flushes

How many times have you experienced this since the accident?

Did you experience this sense of panic before the accident?

Major Depressive Episode

A : Since the accident, have you felt sad, empty or depressed?

A1 : Did you feel empty or sad for most of the day, nearly every day for at least two weeks?
A2 : Since the accident, have you experienced reduced interest or pleasure in your daily activities?

If yes to A1 or A2 :
A3 : During this time, did your weight or appetite change noticeably?

A4 : During this time, did you sleep much more or much less than usual?

A5 : During this time, did you feel physically restless or slowed down?

A6 : During this time, did you feel fatigued or low in energy?

A7 : During this time, did you feel worthless or very guilty?

A8 : During this time, did you feel indecisive or unable to concentrate?

A9 : During this time, did you think about or attempt to kill yourself?

Had there been times before the accident that you felt like this?

Manic Episode

A : Since the accident, have you felt unusually full of life or irritable for at least a week?

If so:
B1 : During this time, did you feel really great about yourself - like you could do anything?

B2 : During this time, did you feel rested after only a few hours of sleep?

B3 : During this time, did you feel more talkative than usual, or pressured to talk more than usual?

B4 : During this time, did you have many ideas, or feel that your thoughts were racing?

B5 : During this time, did you feel easily distracted?

B6 : During this time, were you very active or physically agitated?

B7 : During this time, did you involve yourself in pleasurable activities that had negative consequences (such as a spending spree)?

Had there been times before the accident that you felt like this?

Diagnostic Criterion (DSM-IV)	i. Criterion Met No (1) Yes (2)	ii. Symptom Status NA (1) Retrospective (2) (not within last month) Current (3) (within last month)	iii. Symptom Onset (time post MVA) NA (1) < 2 days (2) 2-28 days (3) 1-3 months (4) >3 months (5) >6 month delay (6)	iv. Symptom Cessation (time post MVA) NA/Current (1) < 2 days (2) 2-28 days (3) 1-3 months (4) >3 months (5) >6 months (6)
Posttraumatic Stress Disorder (PTSD)			NA	NA
A : Both (1) & (2)		NA	NA	NA
A(1) Experienced, witnessed or was confronted with an event (MVA) that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others		NA	NA	NA
A(2) Response involved intense fear, helplessness, or horror		NA	NA	NA
B : Persistently reexperience the accident in one or more of (1) - (5)				
B(1) Recurrent & intrusive distressing recollections of the event, including images, thoughts, or perceptions				
B(2) Recurrent distressing dreams of the event				
B(3) Acting or feeling as if the event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated)				
B(4) Intense psychological distress at exposure to internal or external cues that symbolise or resemble an aspect of the event				
B(5) Physiological reactivity on exposure to internal or external cues that symbolise or resemble an aspect of the event				
C : 3 or more of (1) - (7). (Persistent & not present before trauma)				
C(1) Efforts to avoid thoughts, feelings, or conversations associated with the trauma				
C(2) Efforts to avoid activities, places, or people that arouse recollections of the trauma				
C(3) Inability to recall an important aspect of the trauma				
C(4) Markedly diminished interest or participation in significant activities				
C(5) Feeling of detachment or estrangement from others				
C(6) Restricted range of affect (e.g., unable to have loving feelings)				
C(7) Sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal life span)				

Diagnostic Criterion (DSM-IV)	i. Criterion Met No (1) Yes (2)	ii. Symptom Status NA (1) Retrospective (2) (not within last month) Current (3) (within last month)	iii. Symptom Onset (time post MVA) NA (1) < 2 days (2) 2-28 days (3) 1-3 months (4) >3 months (5) >6 month delay (6)	iv. Symptom Cessation (time post MVA) NA/Current (1) < 2 days (2) 2-28 days (3) 1-3 months (4) >3 months (5) >6 months (6)
D : 2 or more of (1) - (5). (Persistent & not present before trauma)				
D(1) Difficulty falling or staying asleep				
D(2) Irritability or outbursts of anger				
D(3) Difficulty concentrating				
D(4) Hypervigilance				
D(5) Exaggerated startle response				
E : Duration of symptoms in B, C, & D is more than one month			NA	NA
F : The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning				
Specifiers - Acute : Duration of symptoms less than three months			NA	NA
Chronic : Duration of symptoms is three months or more			NA	NA
With Delayed Onset : Onset of symptoms at least 6 months post-trauma			NA	NA
Acute Stress Disorder (ASD)			NA	NA
A : (as above)		NA	NA	NA
B : During or after event, 3 or more of the following :		NA	NA	NA
B(1) Subjective sense of numbing, detachment, or absence of emotional responsiveness				
B(2) Reduction in awareness of surroundings (e.g., "being in a daze")				
B(3) Derealization				
B(4) Depersonalization				
B(5) Dissociative amnesia (inability to recall an important aspect of the trauma)				
C : Persistent reexperiencing of event. At least one of : recurrent images, thoughts, dreams, illusions, flashback episodes, or a sense of reliving the experience, or distress on exposure to reminders of the traumatic event.				
D : Marked avoidance of stimuli that arouse recollections of the trauma (e.g., Thoughts, feelings, conversations, activities, places, people)				

Diagnostic Criterion (DSM-IV)	i. Criterion Met No (1) Yes (2)	ii. Symptom Status NA (1) Retrospective (2) (not within last month) Current (3) (within last month)	iii. Symptom Onset (time post MVA) NA (1) < 2 days (2) 2-28 days (3) 1-3 months (4) >3 months (5) >6 month delay (6)	iv. Symptom Cessation (time post MVA) NA/Current (1) < 2 days (2) 2-28 days (3) 1-3 months (4) >3 months (5) >6 months (6)
E : Marked symptoms of anxiety or increased arousal (e.g., difficulty sleeping, irritability, poor concentration, hypervigilance, exaggerated startle response, motor restlessness)				
F : As above. Includes impairment of ability to pursue some necessary task.				
G : Duration of 2 days to 4 weeks (within 4 weeks of traumatic event)				
H : Not due to substance, GMC, Brief Psychotic Disorder or exacerbation of preexisting disorder.		NA	NA	NA
Adjustment Disorder				
A : Development of emotional or behavioural symptoms in response to identifiable stressor (MVA) within three months (of the accident).				
B : These symptoms or behaviours are clinically significant as evidenced by either :				
B(1) Marked distress that is in excess if what would be expected from exposure to the stressor				
B(2) Significant impairment in social or occupational (academic) functioning				
C : Does not meet criteria for other Axis 1 Disorder.		NA	NA	NA
D : Symptoms do not represent Bereavement.		NA	NA	NA
E : Symptoms do not persist for more than 6 months (post-MVA)				
Specifiers - *Acute : disturbance < 6 months*			NA	NA
Chronic : disturbance > 6 months			NA	NA
309.4 With Depressed Mood			NA	NA
309.24 With Anxiety			NA	NA
309.28 With Mixed Anxiety and Depressed Mood			NA	NA
309.3 With Disturbance of Conduct			NA	NA
309.4 With Mixed Disturbance of Emotions and Conduct			NA	NA
309.9 Unspecified			NA	NA

Diagnostic Criterion (DSM-IV)	i. Criterion Met No (1) Yes (2)	ii. Symptom Status NA (1) Retrospective (2) (not within last month) Current (3) (within last month)	iii. Symptom Onset (time post MVA) NA (1) < 2 days (2) 2-28 days (3) 1-3 months (4) >3 months (5) >6 month delay (6)	iv. Symptom Cessation (time post MVA) NA/Current (1) < 2 days (2) 2-28 days (3) 1-3 months (4) >3 months (5) >6 months (6)
Brief Psychotic Disorder With Marked Stressor (brief reactive psychosis)				
A : Presence of one (or more) of the following symptoms:		NA	NA	NA
A1 : delusions				
A2 : hallucinations				
A3 : disorganised speech (e.g., frequent derailment or incoherence)				
A4 : grossly disorganised or catatonic behaviour				
B : Duration of an episode of the disturbance is at least 1 day but less than 1 month, with eventual full return to premorbid level of functioning.				
C : The disturbance is not better accounted for by other diagnoses, or due to substance use or a GMC.		NA	NA	NA

Panic Attack (post MVA)				
A discrete period of intense fear or discomfort, in which four (or more) of the following symptoms developed abruptly and reached a peak within 10 minutes:				
(1) palpitations, pounding heart, or accelerated heart rate		NA	NA	NA
(2) sweating		NA	NA	NA
(3) trembling or shaking		NA	NA	NA
(4) sensations of shortness of breath or smothering		NA	NA	NA
(5) feeling of choking		NA	NA	NA
(6) chest pain or discomfort		NA	NA	NA
(7) nausea or abdominal distress		NA	NA	NA
(8) feeling dizzy, unsteady, lightheaded or faint		NA	NA	NA
(9) derealization (feelings of unreality or depersonalization (being detached from oneself)		NA	NA	NA
(10) fear of losing control or going crazy		NA	NA	NA
(11) fear of dying		NA	NA	NA
(12) parathesias (numbness or tingling sensations)		NA	NA	NA
(13) chills or hot flushes		NA	NA	NA

Number of panic attacks since the MVA	
Number of panic attacks before the MVA	
Time elapsed between MVA - 1st attack	
Time elapsed between MVA - most recent attack	

Diagnostic Criterion (DSM-IV)	i. Criterion Met No (1) Yes (2)	ii. Symptom Status NA (1) Retrospective (2) (not within last month) Current (3) (within last month)	iii. Symptom Onset (time post MVA) NA (1) < 2 days (2) 2-28 days (3) 1-3 months (4) >3 months (5) >6 month delay (6)	iv. Symptom Cessation (time post MVA) NA/Current (1) < 2 days (2) 2-28 days (3) 1-3 months (4) >3 months (5) >6 months (6)
Major Depressive Episode (post MVA)				
A : Five (or more) of the following symptoms have been present during the same 2-week period and represent a change from previous functioning; at least one of the symptoms is either A1 or A2. (not due to GMC)				
A1 : depressed mood most of the day, nearly every day (e.g., feels sad or empty)				
A2 : markedly diminished interest or pleasure in all, or almost all, activities most of the day, nearly every day				
A3 : significant weight loss when not dieting, or weight gain (e.g., a change of more than 5% of body weight in a month), or decrease or increase in appetite nearly every day.				
A4 : insomnia or hypersomnia nearly every day				
A5 : psychomotor agitation or retardation nearly every day				
A6 : fatigue or loss of energy nearly every day				
A7 : feelings of worthlessness or excessive or inappropriate guilt (which may be delusional) nearly every day (not merely self-reproach or guilt about being sick)				
A8 : diminished ability to think or concentrate or indecisiveness, nearly every day				
A9 : recurrent thoughts of death (not just fear of dying), recurrent suicidal ideation without a specific plan, or a suicide attempt or a specific plan for committing suicide				
B : Does not meet criteria for a Mixed Episode.		NA	NA	NA
C : Causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.				

Diagnostic Criterion (DSM-IV)	i. Criterion Met No (1) Yes (2)	ii. Symptom Status NA (1) Retrospective (2) (not within last month) Current (3) (within last month)	iii. Symptom Onset (time post MVA) NA (1) < 2 days (2) 2-28 days (3) 1-3 months (4) >3 months (5) >6 month delay (6)	iv. Symptom Cessation (time post MVA) NA/Current (1) < 2 days (2) 2-28 days (3) 1-3 months (4) >3 months (5) >6 months (6)
D : Not due to a substance or GMC.		NA	NA	NA
E : Not Bereavement.		NA	NA	NA

History of Episodes before the MVA	
Time elapsed between MVA - episode	

Manic Episode (post MVA)				
A : A distinct period of abnormally and persistently elevated, expansive, or irritable mood, lasting at least 1 week (or any duration if hospitalised)				
B : During the period of mood disturbance, three (or more) of the following symptoms have persisted (four if the mood is only irritable) and have been present to a significant degree:				
B1 : inflated self-esteem or grandiosity				
B2 : decreased need for sleep (e.g., rested after only 3 hours of sleep)				
B3 : more talkative than usual or pressure to keep talking				
B4 : flight of ideas or subjective experience that thoughts are racing				
B5 : distractibility (i.e., attention too easily drawn to unimportant or irrelevant external stimuli)				
B6 : increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation				
B7 : excessive involvement in pleasurable activities that have a high potential for painful consequences (e.g., engaging in unrestrained buying sprees, sexual indiscretions, or foolish business investments)				
C : *Does not meet criteria for a Mixed Episode.*		NA	NA	NA
D : Causes marked impairment in social, occupational, or other important areas of functioning; or necessitates hospitalisation to prevent harm to self or others; or there are psychotic features.				
E : Not due to substance or GMC		NA	NA	NA

History of Episodes before the MVA	
Time elapsed between MVA - episode	

Appendix C-3: Coding of PACI (Holmes, 1997b) symptom criteria results as confirmed by the more recently developed CAPS for DSM-IV (Blake et al., 1998) and ASDI (Bryant, Harvey, Dang, & Sackville, 1998), and associated confirmation of lifetime diagnostic group membership for each participant.

ASD criteria for lifetime diagnosis (DSM-IV; APA, 1994) (X = criteria met)

Note: Participants who were excluded from the guided imagery study are coded with an asterisk.

Code	B	B1	B2	B3	B4	B5	C	D	E	Group
P1			X				X	X	X	PTSD
P2*							X	X	X	PTSD
P3		X	X				X	X	X	PTSD
P4*							X	X	X	PTSD
P5				X			X	X	X	PTSD
P6							X	X	X	PTSD
P7*		X					X	X	X	PTSD
P8				X			X	X	X	PTSD
P9			X				X	X	X	PTSD
P10*							X	X	X	PTSD
P11				X			X	X	X	PTSD
P12*						X	X	X	X	PTSD
P13*							X	X	X	PTSD
P14		X	X				X	X	X	PTSD
P15							X	X	X	PTSD
P16*							X	X	X	PTSD
P17*							X	X	X	PTSD
P18							X	X	X	PTSD
P19*							X	X	X	PTSD
P20*							X	X	X	PTSD
P21							X	X	X	PTSD
P22							X	X	X	PTSD
P23			X				X	X	X	PTSD
P24							X	X	X	PTSD
P25		X	X				X	X	X	PTSD
P26							X	X	X	PTSD
P27							X	X	X	PTSD
P28							X	X	X	PTSD
P29*				X			X	X	X	PTSD
P30							X	X	X	PTSD

Code	B	B1	B2	B3	B4	B5	C	D	E	Group
A1	X	X	X	X	X		X	X	X	ASD
A2	X	X	X	X			X	X	X	ASD
A3*	X	X	X	X	X	X	X	X	X	ASD
A4	X		X	X	X		X	X	X	ASD
A5	X	X	X	X	X		X	X	X	ASD
A6*	X	X		X	X	X	X	X	X	ASD
A7*	X	X	X	X	X	X	X	X	X	ASD
A8	X	X	X	X			X	X	X	ASD
A9	X	X	X		X		X	X	X	ASD
A10	X	X	X	X	X		X	X	X	ASD
A11*	X	X	X	X		X	X	X	X	ASD
A12	X	X	X	X	X		X	X	X	ASD
A13	X	X	X	X	X		X	X	X	ASD
A14	X	X	X	X	X		X	X	X	ASD
A15*	X	X	X	X		X	X	X	X	ASD
A16*	X	X		X	X	X	X	X	X	ASD
A17	X	X	X	X	X		X	X	X	ASD
A18*	X	X	X	X	X	X	X	X	X	ASD
A19	X	X	X		X		X	X	X	ASD
A20*	X	X	X	X		X	X	X	X	ASD
A21	X	X	X	X	X		X	X	X	ASD
A22*	X	X	X	X	X	X	X	X	X	ASD
A23	X	X	X	X	X		X	X	X	ASD
A24	X	X	X	X	X		X	X	X	ASD
S1*						X	X		X	SUB
S2				X			X	X		SUB
S3*							X		X	SUB
S4*						X	X		X	SUB
S5		X					X		X	SUB
S6			X				X	X		SUB
S7*								X		SUB
S8*							X	X		SUB
S9							X		X	SUB
S10		X		X			X			SUB
S11*							X			SUB
S12			X					X		SUB
S13*							X	X	X	SUB
S14							X			SUB
S15								X		SUB
S16			X				X		X	SUB

Code	B	B1	B2	B3	B4	B5	C	D	E	Group
S17*							X			SUB
S18								X	X	SUB
S19			X	X			X			SUB
S20								X		SUB
S21*						X		X	X	SUB
S22								X	X	SUB
S23		X					X		X	SUB
S24*									X	SUB
S25				X			X		X	SUB
S26*							X		X	SUB
S27*							X			SUB
S28			X					X	X	SUB
S29							X		X	SUB

PTSD criteria for lifetime diagnosis (DSM-IV; APA, 1994) (X = criteria met)

Code	B	B 1	B 2	B 3	B 4	B 5	C	C 1	C 2	C 3	C 4	C 5	C 6	C 7	D	D 1	D 2	D 3	D 4	D 5	Group
P1	X	X	X		X	X	X	X	X		X	X			X	X	X		X	X	PTSD
P2*	X	X			X	X	X	X	X		X				X	X	X	X			PTSD
P3	X	X	X		X	X	X			X	X	X			X	X	X		X	X	PTSD
P4*	X	X	X	X	X		X				X		X	X	X	X	X	X			PTSD
P5	X	X	X		X	X	X	X	X	X	X				X	X	X	X			PTSD
P6	X	X	X		X	X	X				X		X	X	X	X		X	X	X	PTSD
P7*	X	X		X	X	X	X				X		X	X	X	X	X	X	X	X	PTSD
P8	X		X		X		X	X	X		X	X			X	X		X	X	X	PTSD
P9	X	X	X	X	X	X	X			X	X	X		X	X	X		X	X	X	PTSD
P10*	X	X					X		X				X	X	X	X	X		X	X	PTSD
P11	X	X	X	X		X	X	X		X	X	X			X	X	X		X		PTSD
P12*	X	X			X	X	X				X	X	X		X	X	X		X		PTSD
P13*	X	X		X		X	X				X		X	X	X	X	X		X	X	PTSD
P14	X		X		X	X	X				X	X	X		X	X	X	X		X	PTSD
P15	X	X		X	X	X	X	X	X	X	X				X	X	X		X		PTSD
P16*	X	X	X	X	X		X				X		X	X	X	X	X	X		X	PTSD
P17*	X	X	X			X	X	X	X		X	X			X	X		X			PTSD
P18	X	X	X		X	X	X	X	X	X		X	X		X	X	X		X		PTSD
P19*	X		X	X	X	X	X	X	X			X	X	X	X	X		X		X	PTSD
P20*	X	X		X		X	X	X				X	X	X	X	X		X			PTSD

Code	B	B 1	B 2	B 3	B 4	B 5	C	C 1	C 2	C 3	C 4	C 5	C 6	C 7	D	D 1	D 2	D 3	D 4	D 5	Group
P21	X	X		X	X	X	X					X	X	X	X	X	X	X	X		PTSD
P22	X		X		X	X	X	X	X		X		X		X	X	X	X			PTSD
P23	X	X	X		X	X	X	X	X		X	X			X	X		X			PTSD
P24	X	X	X		X	X	X	X		X	X			X	X	X	X		X		PTSD
P25	X	X		X	X	X	X	X	X		X	X			X	X	X	X	X	X	PTSD
P26	X		X			X	X	X	X	X	X				X	X	X		X		PTSD
P27	X	X			X	X	X			X	X		X	X	X	X		X			PTSD
P28	X	X	X	X	X		X		X		X	X		X	X	X	X	X	X	X	PTSD
P29*	X	X	X		X		X	X	X		X	X		X	X	X	X	X		X	PTSD
P30	X		X		X	X	X		X		X		X	X	X	X			X	X	PTSD
A1												X									ASD
A2							X	X			X	X									ASD
A3*										X		X									ASD
A4	X	X		X	X	X						X									ASD
A5												X			X	X		X			ASD
A6*										X		X									ASD
A7*										X		X									ASD
A8	X	X	X	X	X							X									ASD
A9												X									ASD
A10							X		X			X	X								ASD
A11*										X		X									ASD
A12																					ASD
A13	X	X	X	X	X																ASD
A14																					ASD
A15*										X		X									ASD
A16*										X		X									ASD
A17																					ASD
A18*										X		X									ASD
A19	X	X		X	X	X						X									ASD
A20*										X		X									ASD
A21												X									ASD
A22*										X		X									ASD
A23												X									ASD
A24												X									ASD
S1*															X	X		X			SUB
S2	X	X	X	X	X	X	X	X	X			X		X							SUB
S3*											X								X	X	SUB
S4*																			X	X	SUB

Code	B	B 1	B 2	B 3	B 4	B 5	C	C 1	C 2	C 3	C 4	C 5	C 6	C 7	D	D 1	D 2	D 3	D 4	D 5	Group
S5	X	X			X	X															SUB
S6	X	X	X		X	X	X	X	X		X			X							SUB
S7*							X	X	X		X		X								SUB
S8*	X	X	X	X	X	X															SUB
S9															X		X	X			SUB
S10	X	X	X																		SUB
S11*	X	X			X	X															SUB
S12	X	X	X	X	X	X	X	X	X			X	X								SUB
S13*															X		X	X			SUB
S14	X	X	X		X	X															SUB
S15							X	X	X		X		X	X							SUB
S16															X		X			X	SUB
S17*	X	X	X		X	X															SUB
S18															X		X		X		SUB
S19	X	X	X	X	X	X															SUB
S20							X	X	X		X		X	X							SUB
S21*															X		X	X			SUB
S22																			X		SUB
S23															X		X			X	SUB
S24*																			X		SUB
S25	X	X			X	X															SUB
S26*																			X		SUB
S27*							X	X	X		X		X	X							SUB
S28																	X				SUB
S29																		X	X		SUB

Appendix C-4: The Coping Strategies Inventory (Tobin, Holroyd, & Reynolds, 1984)

The purpose of this inventory is to look at how people deal with having a serious car accident. Consider each item, and circle the extent to which you used it in dealing with your accident.

		Not at all	A little	Somewhat	Much	Very Much
1.	I just concentrated on what I had to do next.	1	2	3	4	5
2.	I tried to get a new angle on the situation.	1	2	3	4	5
3.	I found ways to blow off steam.	1	2	3	4	5
4.	I accepted sympathy and understanding from someone.	1	2	3	4	5
5.	I slept more than usual.	1	2	3	4	5
6.	I hoped the problem would take care of itself.	1	2	3	4	5
7.	I told myself that if I wasn't so careless, things like this wouldn't happen.	1	2	3	4	5
8.	I tried to keep my feelings to myself.	1	2	3	4	5
9.	I changed something so it would turn out all right.	1	2	3	4	5
10.	I looked for the silver lining, so to speak; tried to look on the bright side of things.	1	2	3	4	5
11.	I did think some things to get it out of my system.	1	2	3	4	5
12.	I found somebody who was a good listener.	1	2	3	4	5
13.	I went along as if nothing were happening.	1	2	3	4	5
14.	I hoped a miracle would happen.	1	2	3	4	5
15.	I realized that I brought the problem on myself.	1	2	3	4	5
16.	I spent more time alone.	1	2	3	4	5
17.	I stood my ground and fought for what I needed.	1	2	3	4	5
18.	I told myself things that helped me feel better.	1	2	3	4	5
19.	I let my emotions go.	1	2	3	4	5
20.	I talked to someone about how I was feeling.	1	2	3	4	5
21.	I tried to forget the whole thing.	1	2	3	4	5
22.	I wished that I never let myself get involved with the situation.	1	2	3	4	5
23.	I blamed myself.	1	2	3	4	5
24.	I avoided my family and friends.	1	2	3	4	5
25.	I made a plan of action and followed it.	1	2	3	4	5
26.	I looked at things in a different light and tried to make the best of what was available.	1	2	3	4	5
27.	I let out my feelings to reduce the stress.	1	2	3	4	5
28.	I just spent more time with people I liked.	1	2	3	4	5
29.	I didn't let it get to me; I refused to think about it too much.	1	2	3	4	5
30.	I wished that the situation would somehow go away.	1	2	3	4	5
31.	I criticized myself for what had happened.	1	2	3	4	5
32.	I avoided being with people.	1	2	3	4	5
33.	I tackled the problem head on.	1	2	3	4	5
34.	I asked myself what was really important, and discovered that things weren't so bad after all.	1	2	3	4	5
35.	I let my feelings out somehow.	1	2	3	4	5
36.	I talked to someone that I was very close to.	1	2	3	4	5
37.	I decided that it was really someone else's problem and not mine.	1	2	3	4	5
38.	I wished that the situation had never started.	1	2	3	4	5

		Not at all	A little	Somewhat	Much	Very Much
39.	Since what happened was my fault, I really chewed myself out.	1	2	3	4	5
40.	I didn't talk to other people about the problem.	1	2	3	4	5
41.	I knew what had to be done, so I doubled my efforts and tried harder to make things work.	1	2	3	4	5
42.	I convinced myself that things aren't quite as bad as they seemed.	1	2	3	4	5
43.	I let my emotions out.	1	2	3	4	5
44.	I let my friends help out.	1	2	3	4	5
45.	I avoided the person who was causing the trouble.	1	2	3	4	5
46.	I had fantasies or wishes about how things might turn out.	1	2	3	4	5
47.	I realized that I was personally responsible for my difficulties and really lectured myself.	1	2	3	4	5
48.	I spent some time by myself.	1	2	3	4	5
49.	It was a tricky problem, so I had to work around the edges to make things come out OK.	1	2	3	4	5
50.	I stepped back from the situation and put things into perspective.	1	2	3	4	5
51.	My feelings were overwhelming and they just exploded.	1	2	3	4	5
52.	I asked a friend or relative I respect for advice.	1	2	3	4	5
53.	I made light of the situation and refused to get too serious about it.	1	2	3	4	5
54.	I hoped that if I waited long enough things would turn out OK.	1	2	3	4	5
55.	I kicked myself for letting this happen.	1	2	3	4	5
56.	I kept my thoughts and feelings to myself.	1	2	3	4	5
57.	I worked on solving the problems in the situation.	1	2	3	4	5
58.	I recognized the way I looked at the situation so things didn't look so bad.	1	2	3	4	5
59.	I got in touch with my feelings and just let them go.	1	2	3	4	5
60.	I spent some time with my friends.	1	2	3	4	5
61.	Every time I thought about it I got upset; so I just stopped thinking about it.	1	2	3	4	5
62.	I wished I could have changed what happened.	1	2	3	4	5
63.	It was my mistake and I needed to suffer the consequences.	1	2	3	4	5
64.	I didn't let my family and friends know what was going on.	1	2	3	4	5
65.	I struggled to resolve the problem.	1	2	3	4	5
66.	I went over the problem again and again in my mind and finally saw things in a different light.	1	2	3	4	5
67.	I was angry and really blew up.	1	2	3	4	5
68.	I talked to someone who was in a similar situation.	1	2	3	4	5
69.	I avoided thinking or doing anything about it.	1	2	3	4	5
70.	I thought about fantastic or unreal things that made me feel better.	1	2	3	4	5
71.	I told myself how stupid I was.	1	2	3	4	5
72.	I did not let others know how I was feeling.	1	2	3	4	5

Scoring Sheet for the Coping Strategies Inventory

Problem-solving	Cognitive-restriction	Express-emotions
1	2	3 .....
9	10	11 .....
17	18	19 .....
25	26	27 .....
33	34	35 .....
41	42	43 .....
49	50	51 .....
57	58	59 .....
65	66	67 .....
Total :	Total :	Total :

Social-support	Problem-avoidance	Wishful-thinking
4	5	6 .....
12	13	14 .....
20	21	22 .....
28	29	30 .....
36	37	38 .....
44	45	46 .....
52	53	54 .....
60	61	62 .....
68	69	70 .....
.....	.....	.....
Total :	Total :	Total :

Self-criticism	Social-withdrawal
7	8 .....
15	16 .....
23	24 .....
31	32 .....
39	40 .....
47	48 .....
55	56 .....
63	64 .....
71	72 .....
Total :	Total :

Appendix C-5: The Beliefs Inventory (Davis, Eshelman, & McKay, 1995)

Below are a number of statements. You should tick "agree" or "disagree" for each statement. It is not necessary to think over any item for very long. Mark your answer quickly and go on to the next statement. Be sure to mark how you actually think about the statement, *not* how you think you *should* think.

	Agree	Disagree	
·	_____	_____	1. It is important to me that others approve of me.
·	_____	_____	2. I hate to fail at anything.
·	_____	_____	3. People who do wrong deserve what they get.
··	_____	_____	4. I usually accept what happens philosophically.
··	_____	_____	5. If a person wants to, he can be happy under almost any circumstances.
·	_____	_____	6. I have a fear of some things that often bothers me.
·	_____	_____	7. I usually put off important decisions.
·	_____	_____	8. Everyone needs someone he can depend on for help and advice.
·	_____	_____	9. "A zebra cannot change his stripes".
·	_____	_____	10. I prefer quiet leisure above all things.
··	_____	_____	11. I like the respect of others, but I don't have to have it.
·	_____	_____	12. I avoid things I cannot do well.
·	_____	_____	13. Too many evil persons escape the punishment they deserve.
··	_____	_____	14. Frustrations don't upset me.
··	_____	_____	15. People are disturbed not by situations but by the view they take of them.
··	_____	_____	16. I feel little anxiety over unexpected dangers or future events.
··	_____	_____	17. I try to go ahead and get irksome tasks behind me when they come up.
·	_____	_____	18. I try to consult an authority in important decisions.
·	_____	_____	19. It is almost impossible to overcome the influences of the past.
··	_____	_____	20. I like to have a lot of irons in the fire.
·	_____	_____	21. I want everyone to like me.
··	_____	_____	22. I don't mind competing in activities in which others are better than I.
·	_____	_____	23. Those who do wrong deserve to be blamed
·	_____	_____	24. Things should be different from the way they are.
··	_____	_____	25. I cause my own moods.
·	_____	_____	26. I often can't get my mind off some concern.
·	_____	_____	27. I avoid facing my problems.
·	_____	_____	28. People need a source of strength outside themselves.
··	_____	_____	29. Just because something once strongly affects your life doesn't mean it need do so in the future.
··	_____	_____	30. I'm most fulfilled when I have lots to do.
··	_____	_____	31. I can like myself even when many others don't.
··	_____	_____	32. I like to succeed at something, but I don't feel I have to.
·	_____	_____	33. Immorality should be strongly punished.
·	_____	_____	34. I often get disturbed over situations I don't like.

	Agree	Disagree	
..	_____	_____	35. People who are miserable have usually made themselves that way.
..	_____	_____	36. If I can't keep something from happening, I don't worry about it.
..	_____	_____	37. I usually make decisions as promptly as I can.
.	_____	_____	38. There are certain people that I depend on greatly.
..	_____	_____	39. People overvalue the influence of the past.
..	_____	_____	40. I most enjoy throwing myself into a creative project.
.	_____	_____	41. If others dislike me, that's their problem, not mine.
.	_____	_____	42. It is highly important to me to be successful in everything I do.
..	_____	_____	43. I seldom blame people for their wrong doings.
..	_____	_____	44. I usually accept things the way they are, even if I don't like them.
..	_____	_____	45. A person won't stay angry or blue long unless he keeps himself that way.
.	_____	_____	46. I can't stand to take chances.
.	_____	_____	47. Life is too short to spend it doing unpleasant tasks.
..	_____	_____	48. I like to stand on my own two feet.
.	_____	_____	49. If I had had different experiences I could be more like I want to be.
.	_____	_____	50. I'd like to retire and quit working entirely.
.	_____	_____	51. I find it hard to go against what others think.
..	_____	_____	52. I enjoy activities for their own sake, no matter how good I am at them.
.	_____	_____	53. The fear of punishment helps people be good.
..	_____	_____	54. If things annoy me, I just ignore them.
.	_____	_____	55. The more problems a person has, the less happy he will be.
..	_____	_____	56. I am seldom anxious over the future.
..	_____	_____	57. I seldom put things off.
..	_____	_____	58. I am the only one who can really understand and face my problems.
..	_____	_____	59. I seldom think of past experiences as affecting me now.
..	_____	_____	60. Too much leisure time is boring.
..	_____	_____	61. Although I like approval, It's not a real need for me.
.	_____	_____	62. It bothers me when others are better than I am at something.
.	_____	_____	63. Everyone is basically good.
..	_____	_____	64. I do what I can to get what I want and then don't worry about it.
..	_____	_____	65. Nothing is upsetting in itself - only in the way you interpret it.
.	_____	_____	66. I worry a lot about certain things in the future.
.	_____	_____	67. It is difficult for me to do unpleasant chores.
..	_____	_____	68. I dislike for others to make my decisions for me.
.	_____	_____	69. We are slaves to our personal histories.
.	_____	_____	70. I sometimes wish I could go to a tropical island and just lie on the beach forever.
.	_____	_____	71. I often worry about how much people approve of and accept me.
.	_____	_____	72. It upsets me to make mistakes.
.	_____	_____	73. It's unfair that "the rain falls on both the just and the unjust".

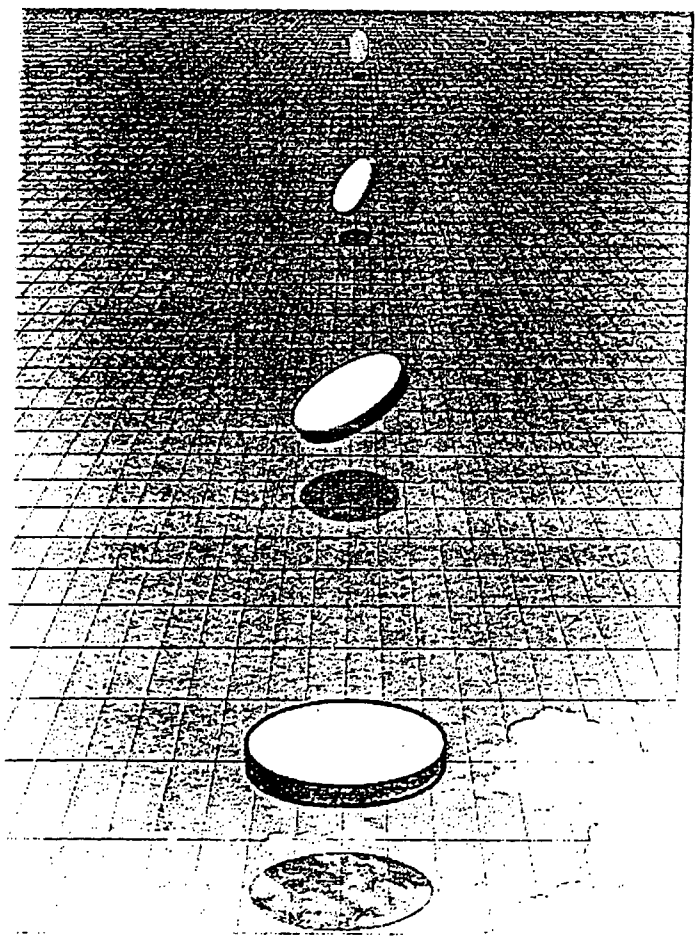
	Agree	Disagree	
..	_____	_____	74. I am fairly easy-going about life.
.	_____	_____	75. More people should face up to the unpleasantness of life.
.	_____	_____	76. Sometimes I can't get a fear off my mind.
..	_____	_____	77. A life of ease is seldom very rewarding.
.	_____	_____	78. I find it easy to seek advice.
.	_____	_____	79. Once something strongly affects your life, it always will.
.	_____	_____	80. I love to lie around.
.	_____	_____	81. I have considerable concern with what people are feeling about me.
.	_____	_____	82. I often become quite annoyed over little things.
..	_____	_____	83. I usually give someone who has wronged me a second chance.
.	_____	_____	84. People are happiest when they have challenges and problems to overcome.
..	_____	_____	85. There is never any reason to remain sorrowful for very long.
..	_____	_____	86. I hardly ever think of such things as death or atomic war.
..	_____	_____	87. I dislike responsibility.
..	_____	_____	88. I dislike having to depend on others.
.	_____	_____	89. People never change basically.
.	_____	_____	90. Most people work too hard and don't get enough rest.
..	_____	_____	91. It is annoying but not upsetting to be criticised.
..	_____	_____	92. I'm not afraid to do things which I cannot do well.
..	_____	_____	93. No one is evil, even though his deeds may be.
..	_____	_____	94. I seldom become upset over the mistakes of others.
..	_____	_____	95. Man makes his own hell within himself.
.	_____	_____	96. I often find myself planning what I would do in different dangerous situations.
..	_____	_____	97. If something is necessary, I do it even if it is unpleasant.
..	_____	_____	98. I've learned not to expect someone else to be very concerned about my welfare.
..	_____	_____	99. I don't look upon the past with any regrets.
.	_____	_____	100. I can't feel really content unless I'm relaxed and doing nothing.

Appendix C-6: Holmes, Williams, and Haines (2001a)

Holmes, G.E., Williams, C.L., & Haines, J. (2001a) Cognitive profiles of Acute Stress Disorder and Posttraumatic Stress Disorder following motor vehicle accident trauma. In R. Roth & S. Neil (Eds.), *A matter of life: Psychological theory, research and practice* (pp.277-285). Lengerich, Germany: Pabst Science.

Roswith Roth, Sandra Neil (Eds.)

A Matter of Life:
Psychological Theory, Research
and Practice



PABST

COGNITIVE PROFILES OF ACUTE STRESS DISORDER AND POSTTRAUMATIC STRESS DISORDER FOLLOWING MOTOR VEHICLE ACCIDENT TRAUMA.

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INTRODUCTION

The conceptualisation of posttraumatic responses as psychiatric entities is formally recognised by the inclusion of Posttraumatic Stress Disorder (PTSD) and Acute Stress Disorder (ASD) in the DSM-IV (American Psychiatric Association [APA], 1994). There are two major differences between the diagnostic criteria for PTSD and ASD. The first is symptom duration. The symptoms of PTSD must last at least one month before a diagnosis can be made. The symptoms of ASD must last at least 2 days and no longer than 4 weeks posttrauma. The second major difference is the emphasis on dissociative phenomena in the diagnostic criteria for ASD. Dissociative phenomena, whether adaptive or maladaptive, may exemplify a cognitive profile which distinguishes the diagnoses of ASD and PTSD (Holmes, Williams & Haines, 1998). In practice, ASD and PTSD are commonly considered on a continuum of symptom severity - an assumption which may be based misleadingly on the DSM-IV duration criteria (Holmes et al., 1998). The inclusion of ASD in the DSM-IV highlights that posttraumatic stress can manifest in more than one presentation. It has been widely discussed that individuals respond to trauma in a broad spectrum of ways, from symptom-free coping to the development of severe and debilitating psychiatric illness significantly reducing quality of life (e.g., O'Brien, 1998).

The DSM-IV definition of a traumatic event is "an event during which a person has experienced, witnessed, or was confronted with actual or threatened death or serious injury, or a threat to the physical integrity of self or others.

The person's response to the event must have involved intense fear, helplessness or horror" (APA, 1994). As the definition describes, it is not only the objective nature of the event in terms of injury or life threat that is important in defining an event's traumatic nature. It is also important to consider and evaluate an individual's interpretation of the event and their psychological responses to the experience. Individual interpretation of traumatic experience is a foundation of differential posttraumatic response, and a reason why not all individuals develop ASD or PTSD following a traumatic experience (e.g., Blanchard et al., 1995; Tunnecliffe & Tunnecliffe, 1997).

Individual interpretation of traumatic experience is based on a complex array of factors. Perception of peritrauma stressors in combination with pretrauma disposition and posttrauma environment are factors enmeshed in the mediation of psychological outcomes. It has been hypothesised that significant mediating factors in the development of posttraumatic psychopathology are posttraumatic cognitive processes. Adaptive cognitive styles have been attributed with powers of inoculation against the development of posttraumatic symptoms, and conversely, maladaptive cognitive styles have been associated with the development and maintenance of posttraumatic symptoms (e.g., van der Kolk, McFarlane & Weisaeth, 1996). The cognitive processes underlying coping strategies therefore are important moderators of life event stress-psychopathology relationships (Hovanitz & Kozora, 1989). Traumatic experience can also be mediated by irrational and inflexible thinking due to the challenging nature of extraordinary experiences. Trauma is widely reported to challenge an individual's existing assumptions, beliefs and views about life and the world. It has been reported that severity of posttraumatic symptoms is associated with cognitive biases and distortion, which may be exemplified by the examination of posttraumatic personal belief systems (Blanchard & Hickling, 1997).

The introduction of the diagnosis of ASD presents a unique opportunity to profile the cognitive styles of individuals with a non-PTSD posttraumatic diagnosis, with the aim of identifying cognitive factors which may be targeted in the early assessment and treatment of posttraumatic responses and be used as prognostic indicators. If cognitive factors can be identified which distinguish individuals diagnosed with ASD from those with PTSD and subclinical diagnoses, ASD as a distinct diagnostic entity may be better understood. Valuable lessons may be learned about the role of adaptive cognitive factors in faster recovery from posttraumatic symptoms.

This paper investigates the cognitive profiles of individuals diagnosed with ASD, PTSD and subclinical responses, using the example of the frequently occurring civilian trauma motor vehicle accidents (MVAs). The cognitive factors that will be examined are the coping strategies used to deal with the MVA trauma, the flexibility and rationality of posttraumatic personal beliefs, and the outcome measure of perceived posttraumatic quality of life. It is hypothesised that the cognitive profile of the PTSD group, reporting the most severe posttraumatic symptoms, will be characterised by greater use of maladaptive

coping strategies, more inflexible posttrauma personal beliefs, and greater posttraumatic dissatisfaction with quality of life, in comparison to the ASD and subclinical groups. It is also hypothesised that the cognitive profile of the ASD group will differ significantly from the profiles of the PTSD and subclinical groups, reflecting the distinct nature of ASD as a diagnostic entity.

METHOD

Participants

Participants were male and female volunteers aged 18 to 75 (N=83). All participants had been involved in a MVA meeting the DSM-IV criteria for a traumatic event, thus each participant had been involved in a life threatening MVA that resulted in intense negative emotions. Each participant was allocated to either the PTSD, ASD or subclinical group (symptom free or symptoms not meeting the criteria for ASD or PTSD), based on responses to a structured clinical interview assessing posttraumatic symptoms following the accident.

Materials

Current posttraumatic symptoms. The Impact of Event Scale (Revised) [IESR] (Weiss & Marmar, 1997) was used to assess posttraumatic symptoms experienced during the past seven days, including the day of testing.
Coping strategies. The Coping Strategies Inventory (CSI) (Tobin, Holroyd & Reynolds, 1984) was used to assess adaptive and maladaptive coping strategies participants used in dealing with MVA trauma.
Posttraumatic personal beliefs. The Beliefs Inventory (BI) (Davis, Eshelman & McKay, 1995) was used to assess posttraumatic personal beliefs. High subscale scores were related to irrationality and inflexibility of personal beliefs.
Posttraumatic perceived quality of life. The Quality of Life Inventory (QOLI) (Frisch, 1994) was used to assess posttraumatic perceived satisfaction with 16 areas of life.
Structured clinical interview. The Post-Accident Clinical Interview (PACI) (Holmes, 1997) is a clinician-administered structured interview designed for this research project, which was used to elicit demographic, peritrauma and diagnostic information.

Procedure

Research volunteers were recruited from the community by way of radio, newspaper and poster advertisements. Volunteers were screened for history of traumatic experiences in an initial interview. The inclusion criterion for participants was involvement in a MVA meeting the DSM-IV criteria for

"trauma". Structured clinical interviews (PACI; Holmes, 1997) were then conducted, during which demographic, peritrauma and diagnostic information was elicited. Debriefing was provided for all participants following discussion of accident details. At the conclusion of each interview, participants were asked to complete a battery of questionnaires, consisting of the IESR, CSI, BI and QOLI. Participants were allocated to three posttraumatic diagnostic groups on the basis of DSM-IV criteria (subclinical, ASD and PTSD). It should be noted that all ASD diagnoses were retrospective, in accordance with the aim of comparing the cognitive profiles of individuals who met the diagnostic criteria for ASD without progression to PTSD, with those individuals with PTSD and subclinical symptoms.

Statistical analysis

Data were collated and analysed using Excel (version 7.0 for Windows '95) and Statistica (version 5.0, 1997 edition). Descriptive statistics for each diagnostic group across the measures were calculated, and one way ANOVA were used to analyse between group main effects. LSD post hoc tests were used to analyse the between group nature of significant main effects. Significance levels were set at .05 for all tests.

RESULTS

Participant characteristics and posttraumatic symptoms

The mean age of the PTSD group (n = 31) was higher than the subclinical (n = 29) and ASD groups (n = 23) (PTSD M = 40.7, SD = 13.7; ASD M = 29.9, SD = 13.8; subclinical M = 31.2, SD = 14.9 years). Each group was comprised of more females than males (PTSD 74.2%, ASD 78.3%, subclinical 62.1% female). In terms of marital status, the majority of the ASD and subclinical groups were single (ASD 65.2%, subclinical 55.2% single), whereas the majority of the PTSD group were married (PTSD 51.6% married). The majority of participants were educated at the tertiary level (PTSD 51.6%, ASD 95.7%, subclinical 86.2% tertiary education). Accident details were comparable between groups, with the majority of participants in each group driving at the time of the accident (PTSD 67.7%, ASD 52.2%, subclinical 65.5% driving), and being physically injured in the accident (PTSD 93.5%, ASD 70.0%, subclinical 69.0% physically injured). Mean time elapsed since the accident was comparable between groups (PTSD M = 84.9, SD = 92.3; ASD M = 76.9, SD = 158.4; subclinical M = 63.9, SD = 87.0 months). With respect to posttraumatic symptoms at the time of testing, there was a significant main effect for IESR total scores to increase with severity of diagnosis ($F(2,80)=15.1$, $p<.0001$). Post hoc analysis demonstrated that the PTSD group scored

significantly higher on the IESR than the subclinical and ASD groups (PTSD M = 32.8, ASD M = 17.0, subclinical M = 9.41). The total scores of the subclinical and ASD groups did not significantly differ from each other, as expected, due to the retrospective nature of the ASD diagnosis.

Coping Strategies

Significant main effects were found for the following subscales: expressing emotions $F(2,80) = 3.55$, $p < .05$; problem avoidance $F(2,80) = 8.80$, $p < .001$; wishful thinking $F(2,80) = 8.11$, $p < .001$; and social withdrawal $F(2,80) = 13.3$, $p < .0001$. Post-hoc analyses found that the ASD group rated the adaptive strategy of expressing emotions significantly higher than the PTSD and subclinical groups (ASD M = 2.58, PTSD M = 2.40, subclinical M = 2.00) [maximum strategy use score = 5.00]. The PTSD group rated the use of the maladaptive strategies problem avoidance (PA), wishful thinking (WT), and social withdrawal (SW) significantly higher than the ASD and subclinical groups (PA: PTSD M = 2.46, ASD M = 2.03, Subclinical M = 1.78; WT: PTSD M = 3.13, ASD M = 2.63, Subclinical M = 2.21; SW : PTSD M = 2.80, ASD M = 1.86, Subclinical M = 1.71). The ratings of the ASD and subclinical groups did not differ significantly from each other on these subscales. There were no significant differences in the reported use of the three other adaptive coping strategies by the three groups (problem solving, cognitive restructuring and social support) or the maladaptive self-criticism strategy. Moderate use of these strategies was reported by all groups.

Posttraumatic Personal Beliefs

Significant main effects were found for belief number six "You should feel fear or anxiety about anything that is unknown, uncertain, or potentially dangerous" $F(2,80) = 5.18$, $p < .01$ and belief number nine "The past has a lot to do with determining the present" $F(2,80) = 5.81$, $p < .01$. Post-hoc analyses showed that the PTSD group rated significantly higher agreement with beliefs six and nine than the ASD and subclinical groups (Belief 6 : PTSD M = 5.87, ASD M = 4.30, subclinical M = 3.59; Belief 9 : PTSD M = 5.16, ASD M = 3.35, subclinical M = 3.41 [maximum rating = 10]). The ASD and subclinical groups did not differ significantly from each other on these dimensions. No significant main effects were found for the other beliefs, which were rated moderately by all groups.

Posttraumatic Perceived Quality of Life

Significant main effects were found for Health $F(2,80) = 5.19$, $p < .01$; Work $F(2,80) = 4.76$, $p < .01$; and Play $F(2,80) = 7.75$, $p < .001$. Post-hoc analyses showed that the PTSD group rated their health and play as significantly less satisfactory than the subclinical and ASD groups. The subclinical and ASD

groups did not differ significantly in their reported satisfaction in these life areas. With regard to work, the ASD and PTSD groups both rated this area of life as significantly less satisfying than the subclinical group. The ratings of the ASD and PTSD groups did not differ significantly from each other on the work dimension. Figure 1 shows these significantly different ratings.

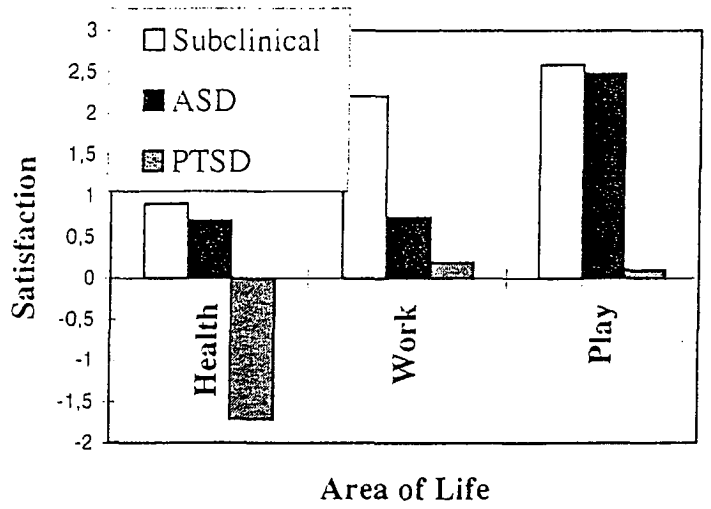

Figure 1: Mean posttraumatic satisfaction scores for areas of life for the three diagnostic groups (score range : -6 to +6).

No significant main effects were found for self-esteem, goals and values, money, learning, creativity, helping, love, friends, children, relatives, home, neighbourhood, community or total quality of life score. These areas of life were rated with degrees of positive satisfaction, with the overall quality of life rating as average for the subclinical and ASD group, and low for the PTSD group.

DISCUSSION

The results showed that the ASD group reported greater use of the adaptive coping strategy 'expressing emotions' than the subclinical and PTSD groups. There were no significant differences in the reported moderate use of the other adaptive coping strategies used by the three groups, which suggests that all three groups used these adaptive coping strategies to a comparable degree in dealing with the trauma. These results demonstrate that the adaptive coping profile of the ASD group was the most positive of the three groups. With respect to the reported use of maladaptive coping strategies, the PTSD group rated significantly greater use of three of the four maladaptive strategies than the ASD and subclinical groups. The reported use of maladaptive strategies was minimal and not significantly different for the ASD and subclinical groups. These results highlight the distinct coping profiles of the three groups, with the ASD group the most adaptive, followed closely by the subclinical group, and the PTSD group reporting significant use of a range of maladaptive coping strategies. Adaptive coping, particularly the expression of emotions, may serve as a mediating factor in the prevention of ASD symptoms progressing to a PTSD diagnosis. In addition, maladaptive coping strategies, as opposed to a deficit in adaptive coping strategies, may be addressed in the treatment of PTSD. An early assessment of coping strategies following MVA trauma may serve as a prognostic indicator.

Between group differences in posttraumatic personal beliefs were evident for two of the ten assessed beliefs. The PTSD group demonstrated greater inflexibility than the ASD and subclinical groups with regard to the beliefs "You should feel fear or anxiety about anything that is unknown, uncertain or potentially dangerous" and "The past has a lot to do with determining the present". These beliefs are commonly associated with generalised fear and anxiety and external locus of control (Davis et al., 1995). These beliefs may be assessed and targeted in the treatment of PTSD. The results suggest that the subclinical and ASD groups were more able to adjust to the experience of motor vehicle accident trauma than the PTSD group, by flexibly integrating the experience into a realistic and rational view of the world. It should also be considered that pretrauma belief systems may have differed between groups and, therefore, had a predisposing effect on posttraumatic psychological outcomes.

The outcome variable, perceived posttraumatic quality of life, demonstrated significant differences in satisfaction between groups in the areas of health, work and play. Health satisfaction referred to being physically fit, not sick, and without pain or disability; work satisfaction referred to career, duties and money earned; and play referred to activities engaged in during free time to relax, have fun or improve the self. The PTSD group was the least satisfied in all three areas. Results indicated that although the ASD group reported less

satisfaction with work than the subclinical group, the ASD profile reflected the maintained ability of the ASD group to "play". This factor may be integral in recovery time, and be an important prognostic indicator and treatment target.
In summary, the cognitive profile of the PTSD group was characterised by greater use of maladaptive coping strategies, as opposed to a deficit in adaptive coping strategy use; more inflexible posttrauma personal beliefs reflecting generalised fear and anxiety and external locus of control; and greater post-traumatic dissatisfaction in the areas of health, work and play than the ASD and subclinical groups. These results support the first hypothesis. In contrast to the cognitive profile of the PTSD group, the profile of the ASD group reflected the most positive coping strategy use of the three groups, in combination with the posttrauma ability to relax, have fun and engage in self-improvement activities. The distinct cognitive profile of the ASD groups supports the second hypothesis, and provides valuable information about the role of adaptive cognitive factors in faster recovery from posttraumatic symptoms. These cognitive profiles highlight potential targets and prognostic indicators for the assessment and treatment of posttraumatic responses and support previous findings on the importance of cognitive factors as moderators of life event stress-psychopathology relationships (e.g., Blanchard & Hickling, 1997; Hovanitz & Kozora, 1989; van der Kolk et al., 1996).

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ACKNOWLEDGEMENTS

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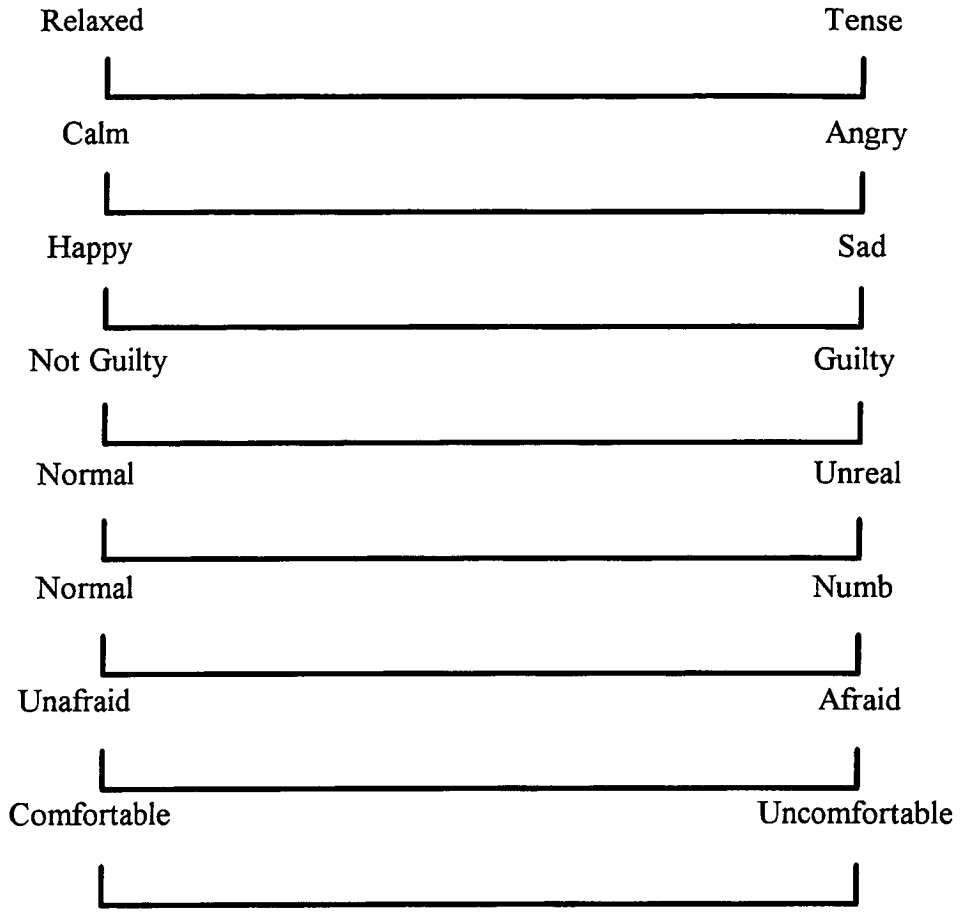
APPENDIX D

ADDITIONAL INFORMATION: CHAPTER SEVEN

Appendix D-1: Visual Analogue Scales

Place a cross on each line scale to describe how you felt during this scene.



How well were you able to put yourself into the scene described?

How close to real life was that scene?

How well were you able to concentrate on imaging the scene?

Appendix D-2: Examples of each guided imagery script type

Names and identifying information have been changed to protect participant anonymity.

Low Arousal Neutral Script

1. Setting the scene

Right, I would like you to remember the time you described to me when you made a cup of tea for yourself at home. You are in your kitchen at home. It is early morning, about 7:15. Really put yourself there. Look around you. See the pitted surface of the floor. See the sunlight coming in the window. See the view of the mountain and the river. You are feeling OK as you stand in the kitchen. Concentrate on that picture right now (pause). You are standing in the kitchen. See the cupboards and u shaped benches. See the white bench tops. See the radio at the end of the bench. See the white goods, and the walk-in pantry. Really put yourself there. See the pale blue color of the walls. The room is very light, warm and sunny. You are feeling OK as you think about making a cup of tea for yourself. Concentrate on that feeling right now (pause). Now open your eyes and switch that scene off.

2. Approach

Right, you are in your kitchen ready to make yourself a cup of tea. Pick up the kettle. Hold the kettle under the tap. Turn on the tap. Really feel the tap in your hand. Put two or three cups of water in the kettle. Feel the weight of the kettle as it fills up. Plug in the kettle to boil the water. Really see the kettle on the bench in front of you. Concentrate on that picture right now (pause). Pour some water into the tea pot to warm it. Look out onto the view. It is a lovely morning. See that the kettle is boiling. Pour the boiling water from the kettle into the tea pot. Really see the steam rise as you pour the boiling water. Pick up a tea spoon. Reach for the herb tea. Put a few tea spoons of herb tea into the tea pot. Concentrate on that picture right now (pause). Now open your eyes and switch that scene off.

3. Incident

Right, you are in your kitchen making some herb tea for yourself. You have placed the tea and water in the tea pot. Put the kettle on again to boil some more water, in case the tea needs to be weakened. You like your tea weak. Leave the herb tea for a few minutes in the tea pot to draw strength. Concentrate on that picture right now (pause). Look around for a mug to have your tea in. Reach for the mug you would like to use. Really feel the weight of the mug in your hand. Really see it there in front of you. Smell the tea as it draws strength. Really breathe in the smell. It is a lovely fragrance. The herb tea is ready. Pour the tea into your mug. Really see the tea as it pours into the mug. You are looking forward to a nice cup of tea. Concentrate on that feeling right now (pause). Now open your eyes and switch that scene off.

4. Consequence

Right, you have poured your cup of tea. See the mug in front of you on the bench. Lift the mug from the bench. Feel the weight of it in your hand as you balance it. You decide to drink the tea in bed as you read the paper. Walk towards your bedroom. See the hallway as you walk to your bedroom. Really picture it. Concentrate on that picture right now (pause). Enter your bedroom. Really see your bedroom. Place your tea on the table beside the bed. Really see the table there. Get back into bed. It is a really pleasant morning. You are feeling quite good, nice and relaxed. You are thinking about what you are doing later in the day. Concentrate on that feeling right now (pause). Now open your eyes and switch that scene off.

High Arousal Neutral Script

1. Setting the scene

Right, I would like you to think back a few years ago when you were water skiing. You are in Queensland. You are in the water at Sunshine Beach. You are wearing your black speedos and a yellow life jacket. You are waist deep in water. You are wearing your water skis. Concentrate on that picture right now (pause). You are behind the speed boat. You are stationary, and waiting for the speed boat to start pulling you along. Really put yourself there behind the boat. The water is warm. Feel the sun on your face. It is a beautiful day. See the blue sky, the sparkling blue water, and the pale yellow sandy beach. Concentrate on that picture right now (pause).Now open your eyes and switch that scene off.

2. Approach

Right, hear the motor of the speed boat starting. Bend your knees and wait for the pull of the line in your hands. Feel the water rush by as you start to stand in the water. You are now upright in the water. Really feel the wind in your wet hair. See the boat making waves in the ocean in front of you. You are now water skiing at a very fast rate. Feel your body glide on the water. Feel your speed almost double as you travel around corners. See your two friends in the boat laughing having a good time. The boat is traveling at high speed. Concentrate on that picture right now (pause). Feel another corner approaching. See ripples from another boat getting larger and crossing your path. This could be tricky. It will be a challenge to stay up. Feel your muscles strain as you try to stay upright. Feel the muscles in your arms pulling tightly on the line to keep you in contact with the boat. You feel warm. Concentrate on that picture right now (pause).Now open your eyes and switch that scene off.

3. Incident

Right, feel the ripples from the boat reach you, becoming larger and rougher. Feel yourself fly. Hang on. You are feeling very relaxed. You know you are at high speed. You are not fighting against it. It is quite rough. Close your eyes tightly as the waves hit your skis. Concentrate on that picture right now (pause). Feel the weight of the water against you. Open your eyes and see the dark, deeper water. Watch the boat turn back towards the beach. Feel your arms start to ache as they hang on tightly. Feel the tightness across your shoulders. The water is helping to keep you cool. Feel it spray up onto your face and chest. Taste the salt water on your lips. Breathe in the sea air. Concentrate on that picture right now (pause).Now open your eyes and switch that scene off.

4. Consequence

Right, feel the boat start to slow down. Soften your grip on the line. It is time to let go. Release your fingers from the line. Let your body flop into the sea. Feel the water around you. Feel your body floating in the life jacket in the warm water. You are really enjoying yourself. Concentrate on that picture right now (pause). You are feeling nice and relaxed. See the boat turning around towards you. See the faces of your friends. They are smiling. Steve wants a turn now. You don't want to get out of the water. It is too nice. Feel the heaviness of your arms and legs in the water after the intense exercise. You feel a little tired, but very refreshed. Concentrate on that picture right now (pause).Now open your eyes and switch that scene off.

Motor Vehicle Accident Script

1. Setting the scene

Right, I would like you to think back to last year, when you were driving from Launceston to Hobart. It is your wife's birthday, September 7th. You are eager to get home and enjoy a bottle of wine and lovely dinner. You are driving on the Midlands Highway. You are about 10 to 15 kilometers outside of Ross. Really put yourself there, behind the wheel. You are feeling relaxed, and looking forward to the evening. Concentrate on that picture right now (pause).You are driving your silver Mazda 626. It has been a busy work trip, but you are glad you got everything done. You feel relieved. Feel the steering wheel in your hands. See the grey dashboard in front of you. It is dusk. See the pink sky and the green paddocks. See the road winding ahead. Concentrate on that picture right now (pause).Now open your eyes and switch that scene off.

2. Approach

Right, see a hill ahead. You cannot see over it. Slow down a little. Drive over the crest of a hill. Drive along the winding road. See a vehicle coming towards you on the other side of the road. See the load it is carrying. It is a four wheel drive, and it has a trailer with a boat on it. Really see the dark coloured car with the white boat. Concentrate on that picture right now (pause). See a red car coming around the side of the boat, on the wrong side of the road. See the car traveling at very high speed towards you. Think, oh my God, it's going to hit me. Quickly look to see if you can swerve to the side of the road. See the electric fence on the paddock. There is no where to go. See the driver of the other car coming towards you. See his wide eyes. See his open mouth. Concentrate on that picture right now (pause). Now open your eyes and switch that scene off.

3. Incident

Right, slam your foot down on the brake. Really feel your foot smash down on the pedal. Hear the screeching brakes. You know it is going to hit. Think of your wife. Think you are going to die on her birthday. Your stomach feels instantly sick. You are screaming "No". Feel the hard impact of the crash. Concentrate on that picture right now (pause). Feel your body being thrown around the vehicle. Think, I am going to die. Hear the smashing of glass. Hear the sounds of metal crunching and objects falling. It feels like everything is happening in slow motion. The car is now stationary. Hear the engine still roaring. You feel terrified. Your body feels trapped. You cannot move. There is no pain. Concentrate on that picture right now (pause). Now open your eyes and switch that scene off.

4. Consequence

Right, you feel no pain. Feel a weird sensation, like you are floating. You can now see your car from above. You can see yourself trapped in the car. See that the bonnet of the car is crushed onto your legs. You feel nothing, like you are detached from it all, like watching a movie. See the other car. See the body of the driver. He has come through the windscreen. His body is face down about two feet away from your face. He is obviously dead. Concentrate on that picture right now (pause). You are still hovering above the scene. You do not know where the car is that was pulling the boat. You can only see the mangled wreck of your car with the other red car. It is hard to tell the two vehicles apart. They seem molded together. See steam rising from the wreck. Hear the engine. It is the only sound you can hear. You feel nothing. Concentrate on that picture right now (pause). Now open your eyes and switch that scene off.

Post Motor Vehicle Accident Script

1. Setting the scene

Right, I would like you to remember what happened at the scene of the car accident. The accident has just happened. You feel no pain. Feel a weird sensation, like you are floating. You can see your car from above. You can see yourself trapped in the car. See that the bonnet of the car is crushed onto your legs. You feel nothing, like you are detached from it all, like watching a movie. Concentrate on that picture right now (pause). You are hovering above the scene. You do not know where the car is that was pulling the boat. You can only see the mangled wreck of your car with the other red car. It is hard to tell the two vehicles apart. They seem molded together. See steam rising from the wreck. Hear the engine. It is the only sound you can hear. You feel nothing. Concentrate on that picture right now (pause). Now open your eyes and switch that scene off.

2. Approach

Right, start to hear other sounds. Hear voices. They seem very far away. You are no longer floating. You are back inside the car. Look around you. See that you are surrounded my metal. It doesn't look like your car at all. Start to feel hot. Feel pins and needles throughout your body. Look at your hands. They are covered in blood. It feels sticky and hot. You cannot move. You are trapped. Concentrate on that picture right now (pause). You cannot believe that you are still alive. You really thought that you were about to die. Feel relief. Feel tears start to fall down your cheeks. You do not feel sad. You feel lucky. It is bizarre. You are sitting in the wreck, thinking how lucky you are. Concentrate on that picture right now (pause).Now open your eyes and switch that scene off.

3. Incident

Right, you are sitting in the car, feeling very strange. Hear people coming towards your car. Hear people screaming. You can hear a woman's voice. She has seen the body. You can see the body too. You feel nothing. You remember the driver's face drove towards you, and now he is lying there, limp. You feel no emotion. It is too horrible to contemplate. Concentrate on that picture right now (pause). It is getting dark. See someone coming towards your car. He seems to be a police officer. He has a calm voice. Hear him tell you that everything will be OK. See him put a blanket over the body in front of you. Tell him, thanks. Others come over to help him. You don't know who they are. See the people pulling at the metal of the wreck to get you out. You feel hot, sticky and numb. Concentrate on that picture right now (pause).Now open your eyes and switch that scene off.

4. Consequence

Right, feel the metal being pulled away from your legs. Feel yourself being lifted out of the car. You feel light-headed, and start to feel a little sick. You are now outside the car. You are being placed on a stretcher. See the ambulance on the side of the road. See the group of people on the side of the road, standing silently. Close your eyes and feel the cool breeze on your face as you are lifted into the ambulance. Concentrate on that picture right now (pause). You are now in the ambulance. Feel a plastic mask being placed on your face. You are lying flat on your back. See the roof of the ambulance. Hear the paramedic tell you to lie very still. Think about your wife and daughters. You cannot wait to see them. See their faces. Close your eyes and picture them at home. Hear the engine of the ambulance start up. You are leaving the scene. Concentrate on that picture right now (pause). Now open your eyes and switch that scene off.

Appendix D-3: FBV (mV) means and standard deviations for each group across the stages of each script ($N = 51$).

Script	Scene		Approach		Incident		Consequence	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
LAN								
PTSD	1.08	(2.47)	1.11	(2.49)	0.20	(2.32)	2.41	(4.57)
ASD	1.69	(4.38)	-0.50	(2.09)	2.63	(6.17)	2.95	(4.03)
Subclinical	0.20	(1.42)	1.16	(6.47)	1.97	(4.68)	2.26	(4.97)
HAN								
PTSD	0.67	(4.42)	0.87	(5.67)	0.06	(2.73)	1.45	(7.12)
ASD	0.85	(3.99)	0.86	(4.41)	-0.59	(4.70)	-0.03	(4.68)
Subclinical	0.53	(4.25)	0.30	(3.04)	-0.79	(4.52)	-2.40	(7.19)
MVA								
PTSD	2.85	(6.58)	3.23	(9.71)	3.08	(9.36)	4.16	(9.44)
ASD	0.42	(0.79)	3.38	(5.70)	0.84	(2.61)	-0.11	(2.63)
Subclinical	-0.62	(8.35)	0.82	(8.12)	-1.14	(7.69)	-1.19	(7.89)
Post MVA								
PTSD	0.74	(4.68)	-1.95	(4.50)	-0.88	(2.06)	-0.45	(2.05)
ASD	0.20	(3.68)	1.62	(2.73)	1.09	(5.69)	0.22	(6.15)
Subclinical	-2.32	(5.03)	-1.46	(4.49)	-0.74	(2.94)	-1.61	(4.45)

Appendix D-4: Post hoc statistics for differences in FBV (mV) mean change scores between groups at each stage of each script (*df* = 2, 48).

Script	Stage	*F*	*p*
LAN	Scene	1.38	n.s.
	Approach	1.64	n.s.
	Incident	2.69	n.s.
	Consequence	0.85	n.s.
HAN	Scene	0.35	n.s.
	Approach	0.38	n.s.
	Incident	0.47	n.s.
	Consequence	1.81	n.s.
MVA	Scene	1.68	n.s.
	Approach	0.54	n.s.
	Incident	1.82	n.s.
	Consequence	3.56	<.05
Post MVA	Scene	2.25	n.s.
	Approach	3.63	<.05
	Incident	1.38	n.s.
	Consequence	0.73	n.s.

Appendix D-5: Post hoc statistics for between script differences in FBV (mV) mean change scores at each stage for each group ($df = 2, 48$).

Group	Stage	*F*	*p*
PTSD	Scene	4.37	<.01
	Approach	2.72	n.s.
	Incident	3.39	<.05
	Consequence	3.63	<.05
ASD	Scene	0.46	n.s.
	Approach	2.03	n.s.
	Incident	0.94	n.s.
	Consequence	1.31	n.s.
Subclinical	Scene	0.86	n.s.
	Approach	0.58	n.s.
	Incident	1.07	n.s.
	Consequence	1.65	n.s .

Appendix D-6: Post hoc statistics for differences in FBV (mV) mean change scores across the stages of each script for each group ($df = 2, 48$).

Group	Stage	*F*	*p*
PTSD	LAN	2.40	n.s.
	HAN	1.17	n.s.
	MVA	2.33	n.s.
	Post MVA	1.52	n.s.
ASD	LAN	2.94	n.s.
	HAN	3.15	n.s.
	MVA	5.25	<.005
	Post MVA	1.38	n.s.
Subclinical	LAN	1.08	n.s.
	HAN	2.49	n.s.
	MVA	3.96	<.05
	Post MVA	0.86	n.s .

Appendix D-7: Standard deviations of mean HR (bpm) for each stage of each script for the total sample ($N = 51$).

Script	Scene	Approach	Incident	Consequence
LAN	10.30	10.68	10.02	10.20
HAN	9.43	10.19	9.12	9.57
MVA	10.70	11.00	10.17	9.76
Post MVA	9.88	9.47	8.87	8.57

Appendix D-8: Means and standard deviations for each group across the stages of each script for HR (bpm) ($N = 51$).

Script	Scene		Approach		Incident		Consequence	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
LAN								
PTSD	73.46	(11.96)	72.79	(11.69)	72.85	(11.14)	73.19	(11.74)
ASD	68.50	(12.88)	68.03	(14.00)	69.15	(12.63)	68.80	(12.61)
Subclinical	70.58	(6.06)	70.23	(6.34)	72.08	(6.29)	71.85	(6.25)
HAN								
PTSD	73.89	(10.73)	74.24	(11.20)	74.79	(10.15)	74.22	(11.27)
ASD	68.84	(11.19)	69.33	(12.95)	70.00	(11.29)	69.06	(10.86)
Subclinical	72.29	(6.38)	72.25	(6.43)	72.99	(5.93)	71.89	(6.57)
MVA								
PTSD	77.88	(12.96)	78.41	(12.54)	78.09	(12.25)	76.63	(11.07)
ASD	72.02	(13.01)	73.47	(14.07)	72.34	(11.90)	71.07	(11.67)
Subclinical	73.94	(6.13)	75.15	(6.38)	75.59	(6.35)	74.10	(6.54)
Post MVA								
PTSD	74.12	(11.08)	74.36	(10.88)	74.30	(11.14)	73.57	(10.52)
ASD	70.57	(12.44)	70.01	(11.59)	68.66	(10.17)	68.78	(9.45)
Subclinical	73.19	(6.13)	73.68	(5.95)	72.69	(5.35)	72.28	(5.73)

Appendix D-9: Means and standard deviations for each group for the baseline period preceding each script for HR (bpm) (*N*=51).

Script	LAN		HAN		MVA		Post MVA	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
PTSD	71.76	(11.65)	72.21	(10.73)	72.63	(11.32)	71.54	(10.41)
ASD	67.98	(13.49)	67.53	(10.61)	69.19	(11.98)	67.67	(12.13)
Subclinical	69.66	(6.64)	70.19	(6.16)	71.68	(5.78)	70.86	(6.62)
Total sample	69.95	(10.78)	70.16	(9.42)	71.30	(9.95)	70.17	(9.85)

ANOVA results:

Between group one way ANOVAs:

LAN baseline $F(2,48) = .51, p > .05$

HAN baseline $F(2,48) = 1.03, p > .05$

MVA baseline $F(2,48) = .51, p > .05$

Post MVA baseline $F(2,48) = .70, p > .05$

Repeated measures ANOVA:

Group main effect $F(2,48) = .69, p > .05$

Script main effect $F(3,144) = 2.42, p > .05$

Group x script interaction $F(6,144) = .42, p > .05$

Appendix D-10: Post hoc statistics for differences in mean HR (bpm) between scripts at each stage for the total sample (*df* = 2, 48).

Stage	*F*	*p*
Scene	10.44	<.0001
Approach	19.85	<.0001
Incident	12.03	<.0001
Consequence	7.37	<.0001

Appendix D-11: Post hoc statistics for differences in mean HR (bpm) across the stages of each script for the total sample (*df* = 2, 48).

Script	*F*	*p*
LAN	2.88	n.s.
HAN	2.60	n.s.
MVA	4.31	<.01
Post MVA	2.58	n.s.

Appendix D-12: Means and standard deviations for each group across the stages of each script for RESP (bpm) (*N*=51).

Script	Scene		Approach		Incident		Consequence	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
LAN								
PTSD	15.32	(4.32)	15.47	(4.06)	15.42	(4.22)	15.26	(3.52)
ASD	17.67	(3.60)	18.00	(3.98)	17.80	(3.80)	18.33	(3.37)
Subclinical	15.50	(2.69)	16.21	(3.50)	15.88	(3.97)	16.24	(3.36)
HAN								
PTSD	14.63	(4.81)	15.95	(4.62)	15.63	(4.43)	15.16	(3.67)
ASD	17.60	(4.37)	19.00	(4.05)	19.67	(5.07)	18.93	(3.96)
Subclinical	15.09	(3.31)	15.94	(3.23)	15.97	(3.06)	15.82	(2.81)
MVA								
PTSD	16.74	(3.60)	16.11	(4.19)	16.79	(4.67)	16.63	(4.30)
ASD	18.20	(4.33)	19.67	(4.30)	19.07	(3.99)	18.53	(4.26)
Subclinical	15.74	(3.31)	16.32	(2.79)	16.03	(2.68)	15.76	(3.31)
Post MVA								
PTSD	14.84	(4.00)	16.21	(4.21)	15.74	(4.59)	15.63	(4.74)
ASD	18.93	(3.96)	20.27	(6.42)	19.93	(6.30)	19.53	(4.17)
Subclinical	15.47	(3.47)	16.32	(3.03)	16.82	(3.23)	15.77	(3.72)

Appendix D-13: Means and standard deviations for each group for the baseline period preceding each script for RESP (bpm) ($N = 51$).

Script	LAN		HAN		MVA		Post MVA	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
PTSD	13.26	(3.83)	14.42	(4.29)	14.05	(3.34)	13.37	(3.09)
ASD	15.53	(4.56)	14.53	(4.88)	15.07	(4.17)	15.13	(4.14)
Subclinical	12.59	(3.66)	12.56	(3.49)	13.47	(2.58)	12.29	(2.54)
Total sample	13.71	(4.11)	13.83	(4.24)	14.16	(3.38)	13.53	(3.41)

ANOVA results:

Between group one way ANOVAs:

LAN baseline $F(2,48) = 2.34, p > .05$

HAN baseline $F(2,48) = .32, p > .05$

MVA baseline $F(2,48) = .90, p > .05$

Post MVA baseline $F(2,48) = 3.02, p > .05$

Repeated measures ANOVA:

Group main effect $F(2,48) = 1.94, p > .05$

Script main effect $F(3,144) = .84, p > .05$

Group x script interaction $F(6,144) = 1.25, p > .05$

Appendix D-14: Means and standard deviations for each group across the stages of each script for EMG (mV) ($N = 51$).

Script	Scene		Approach		Incident		Consequence	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
LAN								
PTSD	734.32	(712.09)	730.21	(713.73)	735.68	(718.52)	731.63	(713.27)
ASD	361.40	(338.92)	356.00	(322.34)	371.67	(330.77)	365.07	(315.46)
Subclin.	452.77	(335.41)	447.77	(323.28)	447.24	(319.35)	446.29	(305.65)
HAN								
PTSD	782.42	(736.77)	757.37	(702.45)	765.90	(719.26)	729.95	(671.51)
ASD	313.53	(288.63)	314.07	(277.96)	314.33	(281.86)	320.60	(281.13)
Subclin.	480.71	(301.14)	473.24	(297.35)	482.82	(292.20)	464.29	(286.71)
MVA								
PTSD	649.63	(478.80)	643.90	(449.10)	647.37	(441.38)	655.58	(478.61)
ASD	388.33	(304.68)	399.80	(299.59)	402.60	(280.13)	429.33	(316.30)
Subclin.	445.12	(296.02)	438.53	(281.40)	442.00	(282.70)	445.88	(284.40)
Post MVA								
PTSD	673.21	(608.37)	661.53	(557.30)	660.42	(573.46)	593.58	(513.82)
ASD	334.73	(274.31)	353.20	(287.96)	349.33	(269.91)	353.60	(292.01)
Subclin.	442.29	(275.05)	446.71	(260.17)	448.29	(260.43)	437.94	(259.06)

Appendix D-15: Means and standard deviations for each group for the baseline period preceding each script for EMG (mV) ($N = 51$).

Script	LAN		HAN		MVA		Post MVA	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
PTSD	758.05	(718.80)	674.26	(673.40)	640.00	(440.52)	621.89	(507.21)
ASD	356.47	(330.82)	310.33	(288.98)	370.07	(307.04)	317.40	(286.33)
Subclinical	461.18	(348.73)	504.18	(310.65)	453.59	(297.75)	440.88	(280.10)
Total sample	540.98	(534.65)	510.53	(489.63)	498.47	(371.31)	472.00	(395.87)

ANOVA results:

Between group one way ANOVAs:

LAN baseline $F(2,48) = 2.84, p > .05$

HAN baseline $F(2,48) = 2.45, p > .05$

MVA baseline $F(2,48) = 2.55, p > .05$

Post MVA baseline $F(2,48) = 2.74, p > .05$

Repeated measures ANOVA:

Group main effect $F(2,48) = 3.20, p = .049$

$\Rightarrow$ LSD post hoc, PTSD > ASD (p =.02).

Script main effect $F(3,144) = .70, p > .05$

Group x script interaction $F(6,144) = .51, p > .05$

Appendix D-16: Means and standard deviations for each group across the stages of each script for SCL (υmho) (*N*=51).

Script	Scene		Approach		Incident		Consequence	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
LAN								
PTSD	7.41	(4.56)	7.39	(4.69)	7.30	(4.57)	7.35	(4.58)
ASD	7.43	(6.03)	7.56	(6.17)	7.34	(6.197)	6.84	(5.86)
Subclinical	7.17	(3.91)	6.90	(3.67)	6.46	(3.71)	6.27	(3.68)
HAN								
PTSD	7.32	(4.43)	7.32	(4.71)	7.50	(4.83)	7.20	(4.59)
ASD	8.01	(5.72)	8.08	(5.96)	8.03	(6.03)	7.51	(5.67)
Subclinical	7.33	(3.86)	7.01	(3.84)	7.39	(4.71)	6.95	(3.73)
MVA								
PTSD	7.47	(4.09)	7.59	(4.06)	7.49	(4.06)	7.24	(3.91)
ASD	7.47	(6.11)	7.66	(6.13)	8.10	(6.32)	7.62	(5.82)
Subclinical	6.72	(2.82)	6.55	(3.09)	6.59	(3.43)	6.33	(3.55)
Post MVA								
PTSD	8.01	(4.51)	8.08	(4.73)	7.86	(4.69)	7.75	(4.55)
ASD	7.95	(5.88)	7.69	(5.54)	7.66	(5.59)	7.57	(5.75)
Subclinical	7.14	(2.77)	6.71	(3.02)	6.65	(3.43)	6.49	(4.02)

Appendix D-17: Means and standard deviations for each group for the baseline period preceding each script for SCL (umho) (N = 51).

Script	LAN		HAN		MVA		Post MVA	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
PTSD	7.38	(4.66)	7.15	(4.17)	6.82	(3.83)	7.44	(4.20)
ASD	7.27	(5.82)	8.41	(6.02)	7.67	(6.38)	8.02	(5.98)
Subclinical	7.92	(3.75)	8.12	(4.19)	7.27	(2.93)	7.84	(3.21)
Total sample	7.54	(4.65)	7.86	(4.71)	7.22	(4.38)	7.74	(4.40)

ANOVA results:

Between group one way ANOVAs:

LAN baseline $F(2,48) = .09, p > .05$

HAN baseline $F(2,48) = .31, p > .05$

MVA baseline $F(2,48) = .15, p > .05$

Post MVA baseline $F(2,48) = .07, p > .05$

Repeated measures ANOVA:

Group main effect $F(2,48) = .04, p > .05$

Script main effect $F(3,144) = 1.94, p > .05$

Group x script interaction $F(6,144) = 1.41, p > .05$

Appendix D-18: Descriptive data and analyses for the VASs controlling for script accuracy, imagery clarity and freedom from distractibility ($N = 51$).

Given that no significant between group, between script or between stage differences were found in the control VAS ratings, only the total sample means and standard deviations for each scale across stages have been selected for presentation. The data showed that the participants reported that they were able to image the scenes clearly, that the script information was highly accurate, and participants reported high levels of freedom from distractibility during the tasks.

Scale	Scene		Approach		Incident		Consequence	
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Imagery clarity	98.90	(6.05)	98.37	(7.05)	98.58	(5.96)	97.53	(2.67)
Script accuracy	99.90	(2.05)	99.37	(4.05)	99.58	(6.96)	98.53	(5.67)
Freedom from distractibility	94.90	(6.05)	93.37	(4.05)	95.58	(6.96)	92.53	(5.67)

Appendix D-19: Means and standard deviations for each group across the stages of each script for the VASs tension, anger, guilt, numbness and unreality (0-100) ($N = 51$).

Dimension								
Script	Scene		Approach		Incident		Consequence	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Relaxed/Tense								
LAN								
PTSD	9.11	(9.68)	7.47	(5.41)	7.16	(6.03)	6.05	(4.81)
ASD	9.93	(12.73)	23.67	(30.80)	21.20	(31.08)	25.93	(30.76)
Subclinical	14.53	(16.13)	9.41	(13.89)	9.47	(12.15)	10.18	(17.13)
HAN								
PTSD	28.42	(29.76)	33.68	(33.28)	64.79	(31.00)	39.47	(29.13)
ASD	30.27	(27.04)	32.40	(29.84)	72.47	(15.38)	36.93	(25.88)
Subclinical	39.29	(29.89)	41.82	(31.92)	47.35	(25.72)	41.47	(29.71)
MVA								
PTSD	23.84	(22.94)	53.90	(20.08)	73.79	(22.31)	74.74	(24.89)
ASD	39.13	(25.65)	55.27	(31.17)	84.53	(17.87)	80.40	(15.01)
Subclinical	40.65	(28.18)	55.47	(24.13)	83.71	(17.43)	78.65	(20.65)

(Table continues...)

(Table continued...)

Dimension								
Script	Scene		Approach		Incident		Consequence	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Post MVA								
PTSD	73.26	(27.13)	60.84	(32.99)	74.05	(25.62)	48.68	(28.01)
ASD	62.93	(25.61)	75.13	(20.15)	79.20	(14.18)	65.20	(26.42)
Subclinical	81.59	(21.00)	82.00	(15.54)	80.94	(15.25)	78.53	(14.38)
Calm/Angry								
LAN								
PTSD	9.05	(9.07)	8.00	(6.00)	6.90	(5.34)	6.47	(4.94)
ASD	7.47	(6.77)	15.20	(20.51)	7.13	(7.51)	8.67	(9.16)
Subclinical	13.41	(14.70)	12.59	(19.81)	12.71	(13.03)	10.29	(14.14)
HAN								
PTSD	21.42	(22.34)	17.26	(20.73)	36.37	(28.85)	22.11	(18.85)
ASD	24.40	(24.29)	16.47	(22.02)	34.73	(28.70)	16.73	(15.94)
Subclinical	15.82	(20.18)	25.53	(27.11)	17.59	(19.57)	27.47	(29.64)
MVA								
PTSD	19.68	(20.77)	27.32	(25.68)	44.84	(28.78)	49.00	(30.84)
ASD	24.73	(22.03)	35.53	(28.14)	57.67	(25.51)	59.60	(25.52)
Subclinical	36.06	(29.91)	42.18	(30.16)	56.00	(25.29)	61.35	(29.04)

(Table continues...)

(Table continued...)

Dimension								
Script	Scene		Approach		Incident		Consequence	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Post MVA								
PTSD	49.58	(34.17)	62.90	(33.68)	63.53	(32.52)	37.47	(28.68)
ASD	54.40	(19.58)	61.40	(13.34)	63.27	(21.03)	50.53	(17.43)
Subclinical	61.29	(18.88)	64.71	(17.84)	67.35	(16.33)	62.12	(18.28)
Not guilty/Guilty								
LAN								
PTSD	6.37	(5.80)	6.74	(5.64)	6.47	(5.79)	6.84	(5.80)
ASD	11.67	(17.94)	10.24	(18.04)	9.33	(17.86)	11.67	(17.17)
Subclinical	10.71	(21.30)	11.00	(22.38)	11.12	(22.61)	10.47	(22.60)
HAN								
PTSD	11.68	(15.28)	10.05	(12.42)	8.47	(8.17)	5.79	(5.98)
ASD	16.47	(18.80)	12.47	(15.91)	9.93	(8.94)	4.93	(4.35)
Subclinical	5.88	(5.62)	6.41	(5.42)	8.12	(8.27)	7.47	(7.06)
MVA								
PTSD	16.53	(15.83)	25.21	(24.10)	36.47	(31.14)	33.00	(29.40)
ASD	28.40	(28.17)	37.07	(31.42)	65.07	(30.70)	73.67	(22.09)
Subclinical	15.94	(22.86)	22.59	(26.77)	48.18	(34.93)	52.82	(39.94)

(Table continues...)

(Table continued...)

Dimension								
Script	Scene		Approach		Incident		Consequence	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Post MVA								
PTSD	52.16	(29.95)	60.47	(32.75)	63.31	(34.44)	58.37	(30.30)
ASD	64.33	(25.17)	71.80	(19.31)	73.80	(17.49)	64.67	(24.91)
Subclinical	57.82	(37.79)	57.88	(38.12)	60.35	(39.91)	57.47	(39.53)
Normal/Numb								
LAN								
PTSD	8.58	(8.44)	7.63	(5.86)	6.32	(5.43)	6.47	(5.54)
ASD	9.67	(10.60)	7.47	(9.69)	8.00	(9.82)	6.40	(8.31)
Subclinical	10.41	(14.65)	12.65	(19.31)	10.71	(15.68)	10.71	(16.16)
HAN								
PTSD	11.95	(10.49)	10.95	(10.78)	28.05	(24.06)	23.53	(19.11)
ASD	15.20	(15.28)	13.33	(15.72)	35.80	(25.70)	30.53	(24.32)
Subclinical	6.94	(5.79)	8.00	(6.01)	7.29	(5.92)	8.35	(8.29)
MVA								
PTSD	17.00	(19.71)	32.63	(32.28)	55.05	(36.00)	61.79	(35.98)
ASD	26.00	(22.33)	46.00	(30.61)	87.33	(11.97)	89.00	(9.96)
Subclinical	21.82	(18.98)	27.47	(21.17)	50.88	(30.88)	55.53	(33.24)

(Table continues...)

(Table continued...)

Dimension								
Script	Scene		Approach		Incident		Consequence	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Post MVA								
PTSD	70.47	(24.71)	71.47	(25.42)	59.95	(33.96)	68.32	(26.16)
ASD	64.93	(31.76)	61.87	(30.15)	69.93	(26.50)	66.73	(34.87)
Subclinical	60.59	(28.85)	61.88	(26.35)	64.35	(25.55)	50.41	(25.49)
Normal/Unreal								
LAN								
PTSD	8.58	(8.44)	7.53	(5.91)	6.05	(5.51)	6.16	(5.48)
ASD	14.87	(16.00)	20.13	(27.96)	18.80	(29.71)	17.60	(29.87)
Subclinical	13.06	(20.80)	13.35	(21.70)	11.77	(18.02)	11.24	(18.32)
HAN								
PTSD	11.32	(10.92)	9.90	(11.37)	19.42	(15.57)	29.93	(31.34)
ASD	15.33	(15.24)	13.13	(15.76)	26.07	(16.25)	36.20	(30.18)
Subclinical	13.47	(15.90)	14.00	(15.31)	12.53	(15.05)	12.29	(15.50)
MVA								
PTSD	10.84	(14.68)	39.11	(41.26)	68.79	(34.19)	68.42	(32.74)
ASD	20.20	(20.06)	43.20	(29.32)	88.73	(10.88)	83.93	(16.68)
Subclinical	25.29	(22.35)	30.29	(22.18)	64.41	(31.68)	59.71	(36.72)

(Table continues...)

(Table continued...)

Dimension								
Script	Scene		Approach		Incident		Consequence	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Post MVA								
PTSD	74.90	(26.05)	62.37	(34.05)	76.58	(26.96)	71.53	(25.67)
ASD	60.67	(27.83)	63.80	(26.37)	63.27	(33.17)	68.07	(33.54)
Subclinical	75.29	(25.83)	66.35	(25.83)	56.41	(30.54)	59.65	(23.74)

Appendix D-20: Post hoc statistics for between group differences in VAS ratings at each stage of each script (*df* = 2, 48).

Dimension	Script	Stage	*F*	*p*
Relaxed/Tense	LAN	Scene	0.87	n.s.
		Approach	2.36	n.s.
		Incident	2.90	n.s.
		Consequence	2.47	n.s.
	HAN	Scene	0.62	n.s.
		Approach	0.40	n.s.
		Incident	5.40	<.05
		Consequence	0.06	n.s.
	MVA	Scene	1.53	n.s.
		Approach	0.01	n.s.
		Incident	1.75	n.s.
		Consequence	0.26	n.s.
	Post MVA	Scene	2.26	n.s.
		Approach	3.47	<.05
		Incident	0.61	n.s.
		Consequence	7.10	<.005

(Table continues...)

(Table continued...)

Dimension	Script	Stage	*F*	*p*
Calm/Angry	LAN	Scene	1.43	n.s.
		Approach	0.48	n.s.
		Incident	1.11	n.s.
		Consequence	0.25	n.s.
	HAN	Scene	0.61	n.s.
		Approach	0.80	n.s.
		Incident	3.06	n.s.
		Consequence	1.10	n.s.
	MVA	Scene	1.77	n.s.
		Approach	1.04	n.s.
		Incident	0.62	n.s.
		Consequence	0.20	n.s.
	Post MVA	Scene	0.93	n.s.
		Approach	0.08	n.s.
		Incident	0.14	n.s.
		Consequence	6.47	<.005

(Table continues...)

(Table continued...)

Dimension	Script	Stage	*F*	*p*
Not guilty/Guilty	LAN	Scene	0.84	n.s.
		Approach	0.92	n.s.
		Incident	1.03	n.s.
		Consequence	0.62	n.s.
	HAN	Scene	2.05	n.s.
		Approach	0.88	n.s.
		Incident	0.11	n.s.
		Consequence	1.78	n.s.
	MVA	Scene	1.41	n.s.
		Approach	1.07	n.s.
		Incident	2.92	n.s.
		Consequence	7.17	<.005
	Post MVA	Scene	0.63	n.s.
		Approach	0.87	n.s.
		Incident	0.74	n.s.
		Consequence	0.23	n.s.

(Table continues...)

(Table continued...)

Dimension	Script	Stage	*F*	*p*
Normal/Numb	LAN	Scene	0.12	n.s.
		Approach	1.17	n.s.
		Incident	0.46	n.s.
		Consequence	0.87	n.s.
	HAN	Scene	1.75	n.s.
		Approach	0.57	n.s.
		Incident	8.90	<.001
		Consequence	5.48	<.01
	MVA	Scene	0.82	n.s.
		Approach	2.04	n.s.
		Incident	6.50	<.005
		Consequence	5.50	<.01
	Post MVA	Scene	0.55	n.s.
		Approach	0.74	n.s.
		Incident	0.49	n.s.
		Consequence	2.04	n.s.

(Table continues...)

(Table continued...)

Dimension	Script	Stage	*F*	*p*
Normal/Unreal	LAN	Scene	0.47	n.s.
		Approach	1.74	n.s.
		Incident	1.70	n.s.
		Consequence	1.32	n.s.
	HAN	Scene	0.21	n.s.
		Approach	0.49	n.s.
		Incident	3.51	<.05
		Consequence	3.63	<.05
	MVA	Scene	2.24	n.s.
		Approach	0.84	n.s.
		Incident	3.08	.054
		Consequence	3.54	<.05
	Post MVA	Scene	1.57	n.s.
		Approach	0.08	n.s.
		Incident	2.10	n.s.
		Consequence	0.86	n.s.

Appendix D-21: Means and standard deviations for each group across the stages of each script for sadness, fear, and discomfort VAS ratings (*N*=51).

Scale								
Script	Scene		Approach		Incident		Consequence	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Happy/Sad								
LAN								
PTSD	8.58	(8.44)	7.63	(5.86)	6.05	(5.51)	6.16	(5.48)
ASD	22.67	(19.37)	20.07	(18.50)	21.13	(18.70)	21.13	(19.87)
Subclinical	18.29	(23.59)	13.47	(20.54)	12.59	(19.12)	12.12	(22.42)
HAN								
PTSD	28.47	(26.74)	28.53	(30.54)	32.26	(28.85)	29.00	(29.93)
ASD	25.47	(23.84)	22.00	(26.57)	28.40	(25.59)	20.73	(26.56)
Subclinical	11.59	(10.04)	12.41	(9.51)	12.71	(13.31)	22.12	(24.43)
MVA								
PTSD	25.84	(20.60)	44.37	(23.86)	67.11	(25.17)	74.58	(21.81)
ASD	33.93	(21.42)	44.93	(21.82)	65.67	(21.00)	72.60	(16.77)
Subclinical	34.88	(27.96)	40.94	(27.05)	67.77	(21.27)	63.77	(25.66)
Post MVA								
PTSD	60.05	(31.21)	60.95	(29.24)	80.63	(15.09)	59.53	(27.76)
ASD	72.33	(19.47)	75.33	(15.29)	81.93	(16.25)	68.07	(17.58)
Subclinical	78.94	(16.46)	71.65	(13.11)	78.35	(18.79)	74.53	(16.01)

(Table continues...)

(Table continued...)

Scale								
Script	Scene		Approach		Incident		Consequence	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Unafraid/Afraid								
LAN								
PTSD	8.58	(8.43)	7.95	(5.75)	6.37	(5.52)	6.26	(5.41)
ASD	7.00	(9.06)	6.67	(9.76)	6.80	(9.76)	6.47	(8.76)
Subclinical	4.94	(5.37)	5.94	(8.86)	4.29	(4.99)	8.77	(13.91)
HAN								
PTSD	7.00	(4.99)	7.90	(5.52)	6.84	(6.34)	7.32	(6.65)
ASD	6.13	(4.96)	6.53	(5.11)	6.13	(7.43)	5.13	(5.48)
Subclinical	13.35	(14.45)	14.06	(12.89)	12.24	(14.60)	17.24	(15.93)
MVA								
PTSD	18.16	(18.75)	52.90	(29.34)	79.32	(19.84)	16.32	(32.07)
ASD	26.67	(26.07)	47.47	(36.31)	72.67	(26.63)	71.13	(24.72)
Subclinical	30.94	(28.31)	37.94	(26.55)	63.94	(31.97)	54.88	(30.53)
Post MVA								
PTSD	60.00	(27.07)	59.74	(28.35)	61.16	(30.02)	60.16	(29.07)
ASD	66.53	(27.06)	67.00	(24.80)	68.87	(28.08)	59.07	(31.43)
Subclinical	66.06	(26.02)	62.06	(19.58)	65.12	(19.99)	59.53	(23.34)

(Table continues...)

(Table continued...)

Scale								
Script	Scene		Approach		Incident		Consequence	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Comfortable/Uncomfortable								
LAN								
PTSD	8.72	(8.54)	7.63	(5.86)	6.05	(5.51)	6.16	(5.48)
ASD	7.13	(8.51)	7.60	(9.32)	7.13	(9.58)	6.00	(7.06)
Subclinical	8.00	(10.80)	6.71	(10.25)	7.63	(5.86)	5.18	(7.72)
HAN								
PTSD	17.84	(16.52)	24.79	(21.10)	48.26	(33.25)	35.42	(32.95)
ASD	22.00	(23.18)	26.47	(23.69)	47.27	(32.09)	30.27	(33.96)
Subclinical	16.00	(16.97)	22.59	(17.79)	28.59	(20.69)	29.00	(29.10)
MVA								
PTSD	28.63	(25.09)	50.68	(20.51)	81.26	(17.86)	86.16	(15.22)
ASD	22.73	(22.10)	46.33	(36.76)	73.53	(17.73)	79.07	(15.79)
Subclinical	29.00	(26.89)	41.24	(24.85)	72.71	(30.00)	66.00	(34.19)
Post MVA								
PTSD	62.74	(25.82)	76.79	(13.11)	77.63	(15.02)	75.63	(14.14)
ASD	68.40	(24.83)	75.80	(22.13)	72.20	(23.36)	63.40	(22.98)
Subclinical	78.71	(17.87)	75.41	(19.08)	76.53	(17.98)	75.06	(18.74)

Appendix D-22: Post hoc statistics for across stage differences in mean VAS ratings for each script (*df* = 2, 48).

Dimension	Stage	*F*	*p*	*LSD*
Happy/Sad	LAN	2.07	n.s.	n.a.
	HAN	1.57	n.s.	n.a.
	MVA	75.52	<.0005	6.22
	Post MVA	10.05	<.0005	5.25
Unafraid/Afraid	LAN	0.68	n.s.	n.a.
	HAN	0.95	n.s.	n.a.
	MVA	53.88	<.0005	8.52
	Post MVA	2.14	n.s.	n.a.
Comfortable/Unc.	LAN	0.72	n.s.	n.a.
	HAN	0.88	n.s.	n.a.
	MVA	74.77	<.0005	6.33
	Post MVA	12.05	<.0005	5.70

APPENDIX E

ADDITIONAL INFORMATION: CHAPTER EIGHT

Appendix E-1: Impact of Event Scale [Revised] (Weiss & Marmar, 1995)

Instructions : Below is a list of difficulties people sometimes have after stressful life events. Please read each item, and then indicate how distressing each difficulty has been for you. ***During the past seven days***, WITH RESPECT TO THE ACCIDENT, how much were you distressed or bothered by these difficulties?

	Not at all	A little bit	Moderately	Quite a bit	Extremely
1. Any reminder brought back feelings about it.	**0**	**1**	**2**	**3**	**4**
2. I had trouble staying asleep.	**0**	**1**	**2**	**3**	**4**
3. Other things kept making me think about it.	**0**	**1**	**2**	**3**	**4**
4. I felt irritable and angry.	**0**	**1**	**2**	**3**	**4**
5. I avoided letting myself get upset when I thought about it or was reminded of it.	**0**	**1**	**2**	**3**	**4**
6. I thought about it when I didn't mean to.	**0**	**1**	**2**	**3**	**4**
7. I felt as if it hadn't happened or wasn't real.	**0**	**1**	**2**	**3**	**4**
8. I stayed away from reminders of it.	**0**	**1**	**2**	**3**	**4**
9. Pictures about it popped into my mind.	**0**	**1**	**2**	**3**	**4**
10. I was jumpy and easily startled.	**0**	**1**	**2**	**3**	**4**
11. I tried not to think about it.	**0**	**1**	**2**	**3**	**4**
12. I was aware that I still had a lot of feelings about it, but I didn't deal with them.	**0**	**1**	**2**	**3**	**4**
13. My feelings about it were kind of numb.	**0**	**1**	**2**	**3**	**4**
14. I found myself acting or feeling like I was back at that time.	**0**	**1**	**2**	**3**	**4**
15. I had trouble falling asleep.	**0**	**1**	**2**	**3**	**4**
16. I had waves of strong feelings about it.	**0**	**1**	**2**	**3**	**4**
17. I tried to remove it from my memory.	**0**	**1**	**2**	**3**	**4**
18. I had trouble concentrating.	**0**	**1**	**2**	**3**	**4**
19. Reminders of it caused me to have physical reactions, such as sweating, trouble breathing, nausea, or a pounding heart.	**0**	**1**	**2**	**3**	**4**
20. I had dreams about it.	**0**	**1**	**2**	**3**	**4**
21. I felt watchful and on-guard.	**0**	**1**	**2**	**3**	**4**
22. I tried not to talk about it.	**0**	**1**	**2**	**3**	**4**

Appendix E-2: Stimulus-Response Inventory of Driving-Related Situations [SRI-DRS] (Holmes, 1995)

Adapted from the Stimulus-Response Inventory of Anxiousness
(Endler, Hunt, & Rosenstein, 1962).

How would you react to the following situations? Circle one number on each scale.

1. You are driving where the accident occurred.

Heart beats faster	Not at all	0	1	2	3	4	Much faster
Muscles become tense	Not at all	0	1	2	3	4	Very tense
Perspire	Not at all	0	1	2	3	4	Perspire heavily
Fingers feel cold or numb	Not at all	0	1	2	3	4	Very cold/numb
Breathing becomes rapid	Not at all	0	1	2	3	4	Very rapid

2. You are the passenger in a car and the driver is speeding at 140km/h.

Heart beats faster	Not at all	0	1	2	3	4	Much faster
Muscles become tense	Not at all	0	1	2	3	4	Very tense
Perspire	Not at all	0	1	2	3	4	Perspire heavily
Fingers feel cold or numb	Not at all	0	1	2	3	4	Very cold/numb
Breathing becomes rapid	Not at all	0	1	2	3	4	Very rapid

3. You are driving when a child runs onto the road in front of you.

Heart beats faster	Not at all	0	1	2	3	4	Much faster
Muscles become tense	Not at all	0	1	2	3	4	Very tense
Perspire	Not at all	0	1	2	3	4	Perspire heavily
Fingers feel cold or numb	Not at all	0	1	2	3	4	Very cold/numb
Breathing becomes rapid	Not at all	0	1	2	3	4	Very rapid

4. You are driving and hear a loud crash behind you.

Heart beats faster	Not at all	0	1	2	3	4	Much faster
Muscles become tense	Not at all	0	1	2	3	4	Very tense
Perspire	Not at all	0	1	2	3	4	Perspire heavily
Fingers feel cold or numb	Not at all	0	1	2	3	4	Very cold/numb
Breathing becomes rapid	Not at all	0	1	2	3	4	Very rapid

5. You are driving along an unfamiliar road in heavy rain.

Heart beats faster	Not at all	0	1	2	3	4	Much faster
Muscles become tense	Not at all	0	1	2	3	4	Very tense
Perspire	Not at all	0	1	2	3	4	Perspire heavily
Fingers feel cold or numb	Not at all	0	1	2	3	4	Very cold/numb
Breathing becomes rapid	Not at all	0	1	2	3	4	Very rapid

Appendix E-3: Holmes, Williams, and Haines (1998i)

Holmes, G.E., Williams, C.L., & Haines, J (1998). Posttraumatic psychopathology following motor vehicle accidents. *Mental Health in Australia: Journal of the Australian National Association for Mental Health*, *47*, 32-38.

Abstract

Motor vehicle accident trauma can result in the development of a wide range of psychological symptoms. This paper examines this range of psychopathology following motor vehicle accident trauma. Individuals who had been involved in motor vehicle accidents meeting the DSM-IV criteria for "trauma" were recruited from the community. Participants included males and females aged from 18 to 78. The participants were interviewed using a structured clinical interview and subsequently completed a psychometric assessment of psychopathology. These assessments showed a range of psychopathology following motor vehicle accident trauma, including posttraumatic stress symptoms, depression and anxiety. These symptoms are discussed in terms of the diagnosis and treatment of posttraumatic responses to motor vehicle accident trauma.

A traumatic event has been defined as an event during which an individual is exposed to actual or threatened death or serious injury, or a threat to the physical integrity of self or others (American Psychiatric Association [APA], 1994). In order to meet the DSM-IV criteria for a traumatic event, the individual's response to the event must have involved intense fear, helplessness or horror. An individual's consequent response to the event may be a return to pretrauma functioning, or result in the development of psychopathology.

The conceptualisation of posttraumatic responses as psychiatric entities has been formally recognised by the inclusion of Posttraumatic Stress Disorder (PTSD) and Acute Stress Disorder (ASD) in the DSM-IV (APA, 1994). ASD and PTSD are clinical disorders characterized by a range of symptoms including behavioural avoidance of stimuli associated with the trauma, cognitive reexperiencing of the trauma through intrusive thoughts or nightmares, and increased physiological arousal. These disorders may develop after traumatic events such as combat, natural disaster or civilian trauma, such as motor vehicle accidents. The inclusion of ASD as a diagnostic entity in the DSM-IV has provided support to the recognition that posttraumatic response is not an 'all-or-none' dichotomy. This new diagnostic category has provided recognition that individuals exposed to trauma may experience significant short term

posttraumatic symptoms, and also highlighted for the first time the importance of dissociative symptoms in the framework of posttraumatic psychopathology (Holmes, Williams, & Haines, 1998a).

Historically, posttraumatic psychological symptoms have been considered to result from constitutional vulnerability or genetic predisposition for neurosis (Yehuda & McFarlane, 1995). The development of diagnostic criteria for clinical posttraumatic responses has shifted the recognised aetiology of such disorders from pre-morbid characteristics and vulnerability, to the nature and intensity of the trauma (McGorry, 1995).

Central to investigations of posttraumatic psychological responses is the search for an explanation of why some individuals exposed to a traumatic event develop long term and debilitating psychiatric illnesses, whereas other individuals exposed to the same traumatic event demonstrate few, if any, adverse effects. The common link between existing theoretical models of the development of posttraumatic psychopathology is that they are based largely on cognitive, behavioural and psychophysiological variables. The link between thoughts, actions and bodily responses has been the basis for a number of recent models explaining the theoretical basis of posttraumatic clinical presentations (e.g., Creamer, 1993; McGorry, 1995; Van der Kolk, McFarlane, & Weisaeth, 1996; Wilson & Keane, 1997).

Posttraumatic psychiatric diagnoses are unique in that they result from exposure to a traumatic event. This unique characteristic seems to attract skepticism from some members of the community, who ask the question: How can one life event produce psychopathology? The answer to this question is complex, and thorough discussion of this topic is not possible within the scope of this paper. It can be said, however, that the physical and psychological demands of a traumatic incident affect central nervous system functioning. These effects can be transient or long term (Matsakis, 1992; O'Brien, 1998). Aside from the physical aspects of altered nervous system function, there are many psychological aspects of trauma exposure that may have a lasting impact. For example, exposure to a near death experience, loss of control, survivor guilt and bereavement, traumatic memories, life changes resulting from physical injury, chronic pain and challenges to existing beliefs of personal safety and invulnerability are some of the factors which have been reported to affect psychological recovery from trauma. These factors may play an important role in the development and maintenance of posttraumatic psychopathology (Figley, Bride, & Mazza, 1997; Holmes, Williams, & Haines, 1998b).

In addition to the formal posttraumatic diagnoses of PTSD and ASD, psychopathology which has been associated with posttraumatic responses include panic attacks, depression, substance related disorders, and somatoform disorders (e.g., Goldberg & Gara, 1990; O'Brien, 1998; Scott & Stradling, 1995). These associated conditions may develop in the presence or absence of diagnoses of PTSD or ASD following MVA trauma. It is not uncommon for individuals diagnosed with PTSD to also meet criteria for one or more other DSM-IV axis 1 disorders. This severe deterioration in mental health is indicative of the potentially life changing and debilitating effects of trauma exposure.

It is particularly important in the understanding of posttraumatic disorders and associated psychopathology that responses to a wide range of trauma types are investigated. Until recently, the body of research in the area of posttraumatic reactions focused predominantly on war veterans (e.g., Healy, 1993; Marshall, 1995). Limiting the majority of research to one population restricts the generalisation of findings to other groups. Research has more recently expanded into the areas of civilian trauma types such as motor vehicle accidents and physical assault (e.g., Blanchard et al., 1996; Hickling & Blanchard, 1992; Holmes, Williams, & Haines, 1998c; Watts, 1995). This shift in focus is particularly important as civilian trauma results in considerable psychological, physical and financial costs within the community.

Motor Vehicle Accidents (MVAs) constitute a frequently occurring trauma type. The common nature of MVAs can exclude these events as worthy of consideration as traumatic events. This factor can be a hindrance to early intervention and appropriate mental health care. As reported by Blanchard and Hickling (1997), MVAs are the most frequent trauma type experienced by American men, and have been found to be the single leading cause of PTSD in the general population of the United States of America. Assessment of the prevalence and severity of posttraumatic responses to MVA trauma facilitates increased community awareness of the level of need for psychological support following MVA trauma, and may prevent chronic outcomes by early intervention and prognostic profiling.

The aims of this study are to examine the nature of posttraumatic symptoms and associated psychopathology following MVA trauma in individuals free from pretrauma psychopathology; and to identify specific targets for assessment, diagnosis and treatment of psychopathology following MVA trauma. It is hypothesized that MVA trauma can result in the development of the full spectrum of posttraumatic symptoms and associated psychopathology.

Method

Participants

The participants in this study were 64 males and females aged from 18 to 78 recruited from the community in Tasmania. All participants had been exposed to a motor vehicle accident meeting the DSM-IV criteria for "trauma", and were either drivers, passengers or pedestrians at the time of the accident. The mean time elapsed since the accident was 28 months. Participants were divided into two groups on the basis of posttraumatic diagnosis: Non-PTSD (n=32) and PTSD (n=32). The non-PTSD group consisted of 11 males and 21 females with a mean age of 33.0 years. The PTSD group consisted of 9 males and 23 females with a mean age of 40.1 years.

There were no significant differences between the groups in terms of the objective seriousness of the MVAs. Objective seriousness was determined by the number of vehicles involved, the nature of the physical injuries sustained, number of fatalities, severity of vehicle damage, and objective threat to life. Subjectively, all participants rated the MVA they had experienced as a traumatic event, according to DSM-IV criteria.

Measures

Participants were formally interviewed by the investigator using a structured clinical interview based on DSM-IV criteria. This interview gathered information regarding demographics, accident information, posttraumatic symptoms and pre and posttrauma psychopathology and health. Each participant was then psychometrically assessed using the following battery of questionnaires.

Impact of Event Scale (Revised) (IES-R). The IES-R (Weiss & Marmar, 1997) is a 22-item self-report questionnaire that has been used widely to assess current posttraumatic symptomatology. The questionnaire has three subscales relating to three posttraumatic symptom clusters: intrusive symptoms, avoidance symptoms and hyperarousal symptoms.

Trauma Symptom Inventory (TSI). The TSI (Briere, 1995) is a 100-item self-report questionnaire that has been used widely to assess posttraumatic symptoms experienced during the previous six months. The scale has ten clinical dimensions.

Personality Assessment Inventory (PAI). The PAI (Morey, 1991) is a 344-item self-report questionnaire that has been widely used to assess adult personality variables. The inventory contains four validity scales, eleven clinical scales, five treatment scales, and two interpersonal scales. Full clinical scales are divided into subscales in order for individual constructs to be compared.

Beck Depression Inventory (BDI). The BDI (Beck, Rush, Shaw, & Emery, 1979) is a widely used 21 item questionnaire designed to assess current symptoms of depression. The questionnaire has two subscales relating to cognitive-affective and physiological symptoms of depression respectively.

Beck Anxiety Inventory (BAI). The BAI (Beck, Epstein, Brown, & Steer, 1988) is a widely used 21 item questionnaire designed to assess current symptoms of anxiety. The questionnaire has four symptom cluster subscales relating to neurophysiological, subjective, panic and autonomic symptoms of anxiety.

Procedure

Participants were recruited from the community by poster, newspaper and radio advertisements. Participants were formally interviewed by the investigator and subsequently asked to complete the battery of psychometric tests. Participants were divided into two groups on the basis of posttraumatic diagnoses derived from the structured clinical interview (non-PTSD and PTSD). As this study is part of a larger scale research project, the participants in this study were screened and selected from a wider sample (N=107). Selection was made on the basis of current posttraumatic diagnosis, freedom from pretrauma psychopathology and health problems, and freedom from multiple-trauma contamination. Data were collated and analysed using Excel (version 7.0 for Windows '95) and Statistica (version 5.0, 1997 edition).

Results

The mean scores of the non-PTSD group were compared with those of the PTSD group using independent t-tests for each of the relevant subscales of the psychometric measures.

Posttraumatic symptoms

The mean scores of the PTSD group were significantly higher than the non-PTSD group on all subscales of the IES-R (Intrusive, t (64) = 5.01, p <.0001; Avoidance, t (64) = 3.73, p <.001; Hyperarousal, t (64) = 4.36, p <.0001) and all posttraumatic symptom subscales of the TSI (Anxious Arousal, t (64) = 3.45, p <.001; Intrusive Experiences, t (64) = 3.80, p <.001; Defensive Avoidance, t (64) = 3.79, p <.001; Dissociation, t (64) = 3.28, p <.001). The mean scores of the PTSD group on the above subscales were all clinically significant. Non-PTSD group means were not clinically significant. The means and standard deviations are shown in Table 1.

Table 1.

Means and standard deviations of posttraumatic symptom subscale scores on the IES-R and TSI for the PTSD (n = 32) and non-PTSD (n = 32) groups.

Measure	Non-PTSD		PTSD	
	M	SD	M	SD
IES-R				
Intrusive	4.81	5.28	14.1	9.01
Avoidance	4.09	5.04	10.3	7.90
Hyperarousal	3.09	4.29	10.1	6.60
TSI				
Anxious Arousal	52.0	9.73	60.6	10.4
Intrusive Experiences	51.6	8.07	61.0	11.5
Defensive Avoidance	49.9	8.10	60.1	11.3
Dissociation	54.1	11.1	62.9	10.5

Anxiety and Stress

The mean scores of the PTSD group were significantly higher than those of the non-PTSD group on all subscales of the BAI (Neurophysiological, t (64) = 3.06, p <.001; Subjective, t (64) = 2.64, p <.01; Panic, t (64) = 2.68, p <.01; Autonomic, t (64) = 3.41, p <.001). The mean scores of the PTSD group were also significantly higher than those of the non-PTSD group on the following subscales of the PAI: Anxiety (ANX), t (64) = 6.70, p <.0001; Anxiety-Related Disorders (ARD) t (64) = 7.11, p <.0001; and Stress (STR), t (64) = 2.69, p <.01. The mean scores of the PTSD group on the above subscales were all clinically significant. Non-PTSD group means were not clinically significant. The means and standard deviations are shown in Table 2.

Table 2.

Means and standard deviations of anxiety and stress subscale scores on the BAI and PAI for the PTSD (n = 32) and non-PTSD (n = 32) groups.

Measure	Non-PTSD		PTSD	
	M	SD	M	SD
BAI				
Neurophysiological	1.50	2.00	3.63	3.39
Subjective	2.63	3.44	4.94	3.57
Panic	0.44	0.72	1.53	2.20
Autonomic	1.59	1.60	3.66	3.02
PAI				
Anxiety	49.7	9.77	66.3	10.1
Anxiety-Related Disorders	46.9	9.08	63.6	9.68
Stress	52.8	10.6	62.1	16.4

Depression, somatic complaints and substance-related problems

The mean scores of the PTSD group were significantly higher than those of the non-PTSD group on both subscales of the BDI (Cognitive-affective, t (64) = 3.13, p <.01; and

Physiological, t (64) = 3.28, p <.01). The mean scores of the PTSD group were also significantly higher than those of the non-PTSD group on the following subscales of the PAI: Depression (DEP), t (64) = 7.06, p <.0001; Somatic Complaints (SOM), t (64) = 6.13, p <.0001; Alcohol Problems (ALC), t (64) = 2.08, p <.05; and Drug Problems (DRG), t (64) = 6.15, p <.0001. The mean scores of the PTSD group on the above subscales were all clinically significant. Non-PTSD group means were not clinically significant. The means and standard deviations are shown in Table 3.

Table 3.

Means and standard deviations of associated psychopathology subscale scores on the BDI and PAI for the PTSD (n = 32) and non-PTSD (n = 32) groups.

Measure	Non-PTSD		PTSD	
	M	SD	M	SD
BDI				
Cognitive-affective	4.47	4.96	10.1	8.89
Physiological	3.78	3.06	7.00	4.64
PAI				
Depression	49.2	7.46	73.9	18.4
Somatic Complaints	51.5	9.99	70.9	14.0
Alcohol Problems	51.2	8.36	60.5	23.8
Drug Problems	48.9	6.96	60.8	10.5

PAI profiles

Figure 1 shows the PAI mean t-score profiles of the PTSD and non-PTSD groups of the clinical dimensions referred to by the above t-test results. The horizontal line at the t-score of 59 shows the cut off score above which t-scores are considered to be clinically significant (Morey, 1991).

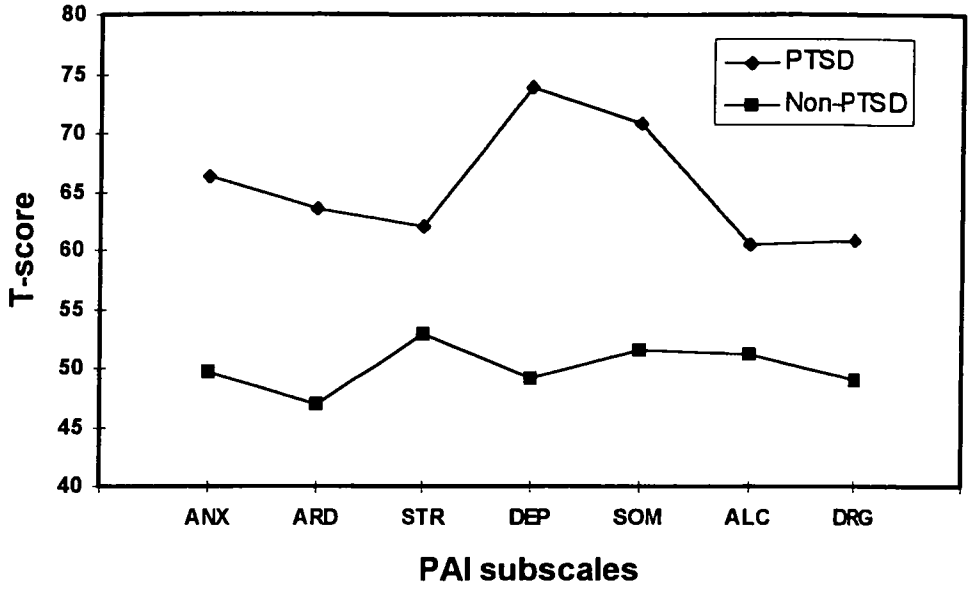


Figure 1.

Mean t-scores of the PTSD (n =32) and non-PTSD (n =32) groups on clinical subscales of the PAI.

Discussion

The mean scores of the PTSD group were significantly higher than those of the non-PTSD group on all subscales of the IES-R, and on all posttraumatic symptoms subscales of the TSI. The PTSD group obtained elevated scores on all posttraumatic symptom dimensions including intrusive, avoidance, hyperarousal, and dissociative symptoms. These findings support the PTSD and non-PTSD group classifications, and highlight the within population variability in psychological sequelae to MVA trauma. It is apparent from the results that some individuals exposed to MVA trauma develop a wide range of clinically significant posttraumatic symptoms whereas other individuals exposed to objectively and subjectively comparable MVA trauma demonstrate few, if any, adverse effects.

With regard to anxiety and stress, the PTSD group reported significantly higher anxiety and stress symptoms than the non-PTSD group. This would be expected as PTSD is an anxiety disorder. The important aspect of this finding is that the PTSD group reported significant manifestations of anxiety in each of the symptom clusters, that is, cognitive, affective and physiological. This finding reinforces existing theory that the treatment approaches to PTSD need to target cognitive, behavioural and psychophysiological variables (e.g., Creamer, 1993; Holmes, Williams, & Haines, 1998c; Wilson & Keane, 1997).

In terms of associated psychopathology, the PTSD group reported significantly higher levels of depression, somatic complaints and substance use related problems than the non-PTSD group. In concordance with the full spectrum of anxiety symptoms, the symptoms of depression reported by the PTSD group encompassed cognitive, affective and physiological manifestations, again reinforcing the importance of multi-target posttrauma treatment. The somatic complaints reported by the PTSD group included significant levels of conversion and somatization symptoms in combination with significant health concerns. It should be reiterated here that the reported pre trauma health and the objectively rated physical injuries sustained by the PTSD group were not significantly worse than those of the non-PTSD group. Somatic complaints are often associated with depression and anxiety (Morey, 1991). The level of substance related problems reported by the PTSD group indicated regular substance use (including alcohol, prescription and illicit drugs) resulting in some adverse consequences. This finding highlights the need for assessing posttrauma coping strategies, and early intervention for the prevention of potentially adverse consequences associated with substance use. It should be noted that not all of the PTSD group demonstrated the same profiles in terms of associated psychopathology. The results described a range of problems which are commonly but not always co-morbid with PTSD.

These findings regarding psychopathology associated with posttraumatic responses support the existing literature regarding PTSD and DSM-IV axis 1 comorbidity (e.g., O'Brien, 1998; Scott & Stradling, 1995), and may also reflect a degree of overlap in diagnostic criteria for these disorders. These findings have demonstrated that posttraumatic responses to MVA trauma vary from non-psychopathological adjustment to the extreme of the development of PTSD and associated psychopathology. These findings reinforce the status of MVA trauma as a significant stressor which can result in the development of psychopathology in individuals free from pre trauma psychopathology.

The results support the hypothesis that MVA trauma can result in the development of the full range of posttraumatic symptoms and associated psychopathology. It should be noted that posttraumatic responses are not always negative in terms of mental health. Positive outcomes can include increased appreciation and enthusiasm for life resulting from awareness of vulnerability (Holmes, Williams, & Haines, 1998b). This may be reflected by the psychopathology free non-PTSD group. The frequency of MVA trauma in Australia reinforces the community need for adequate posttrauma assessment, diagnosis and treatment of posttraumatic responses in affected individuals in order to reduce chronic outcomes and facilitate recovery.

Acknowledgments

We gratefully acknowledge that the Motor Accidents Insurance Board's Injury Prevention and Management Foundation is currently funding this Tasmanian research project which is investigating the psychological effects of exposure to motor vehicle accident trauma.

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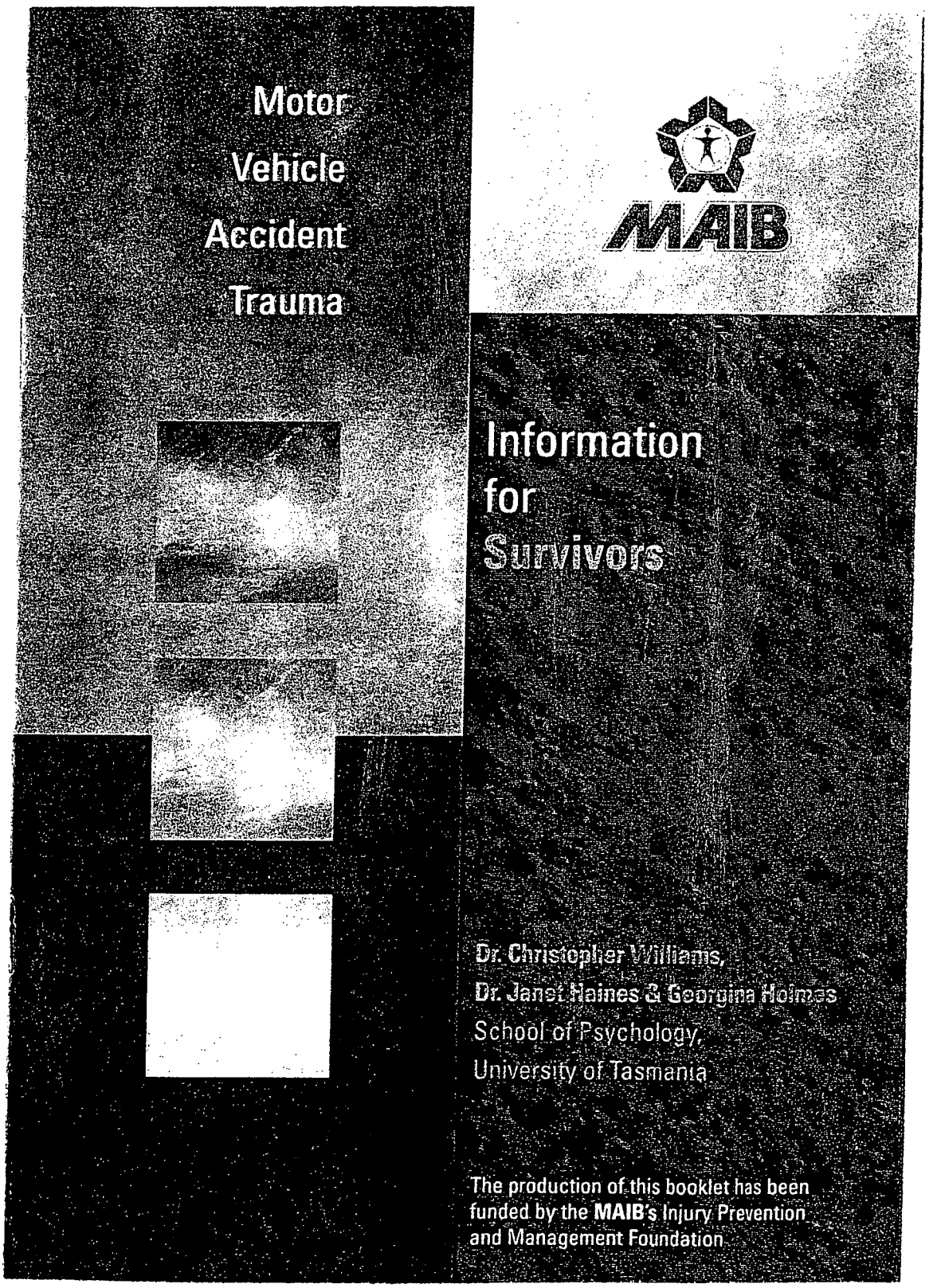
APPENDIX F

ADDITIONAL INFORMATION: CHAPTER NINE

Appendix F-1: Information handbook designed for motor vehicle accident survivors



CONTENTS

About this booklet

Research in the School of Psychology at the University of Tasmania has been investigating psychological responses to motor vehicle accident trauma since 1995. This research has been internationally recognised, and supported by the MAIB's Injury Prevention and Management Foundation. This booklet has been produced by the research team for distribution by MAIB within the Tasmanian community.

Motor Vehicle Accident Trauma

What is a traumatic MVA?

It may be defined as a motor vehicle accident which involves an actual or perceived threat to physical safety, resulting in intense negative emotions such as fear, helplessness or horror for the person involved.

Is MVA trauma common in Tasmania?

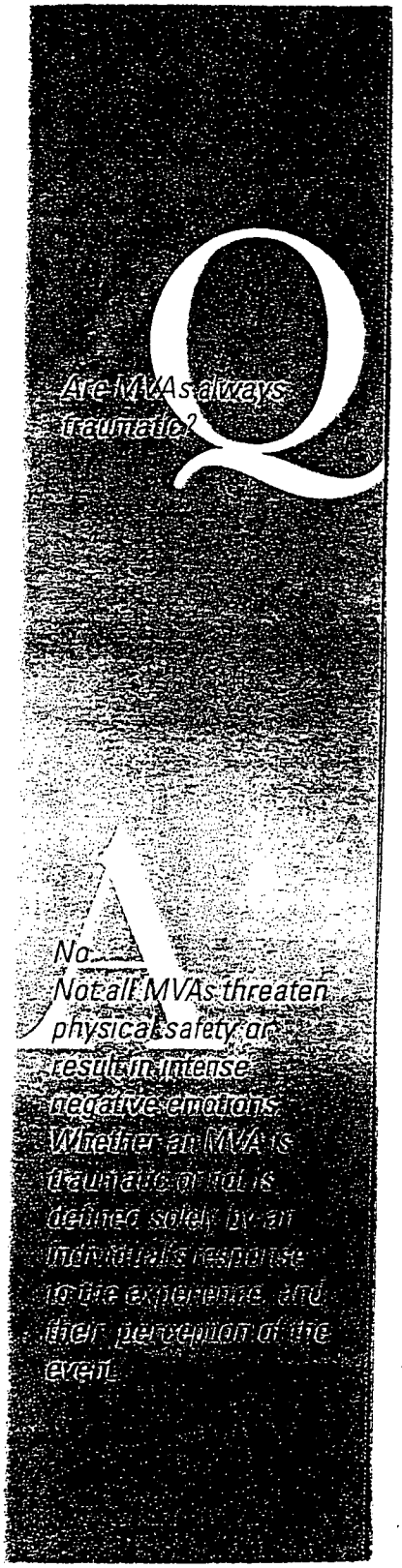
Unfortunately, yes. In addition to frequently occurring fatalities, as indicated by the road toll, less destructive accidents can result in traumatic responses. The common nature of MVA trauma can sometimes result in psychological recovery being overlooked for intervention.

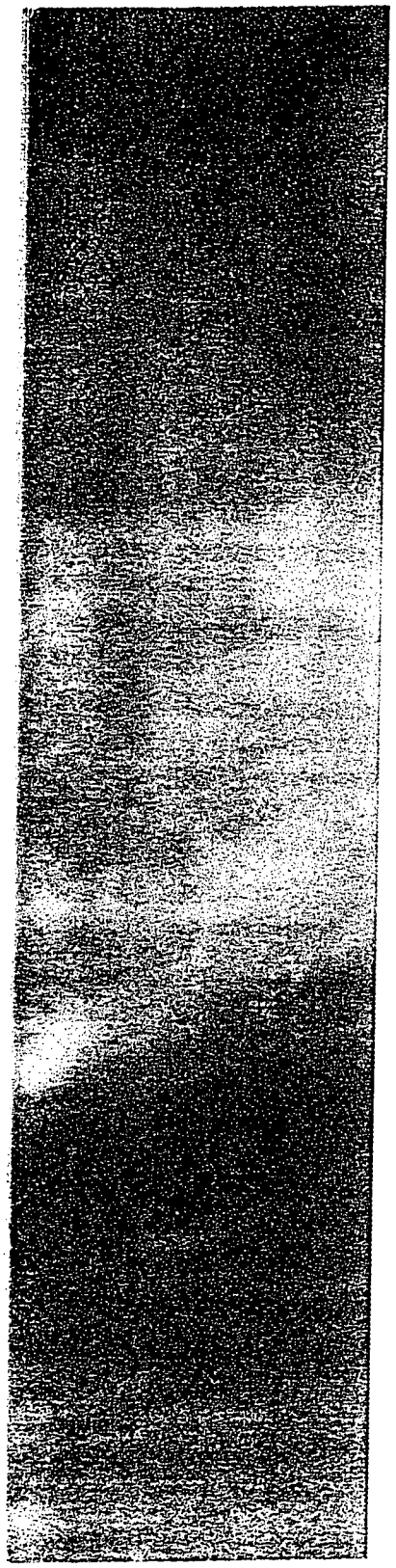
Are MVAs more traumatic for drivers or passengers?

People perceive and respond differently to trauma. Drivers, passengers, pedestrians or witnesses involved in the same accident will respond in their own way. The level of trauma experienced is not solely defined by a person's role in the accident.

Is the car fixed yet?

Often the first priorities after an MVA are medical treatment for physical injuries and dealing with insurance or car repair companies. Initially, psychological intervention for trauma is not a high priority, and in some cases not even considered.





Psychological Effects

How long does psychological trauma last?

Psychological trauma can last from hours to years. Being aware of how the accident has affected you and your life is the first step in assessing whether you should seek psychological intervention.

Adults

The effects of psychological trauma may be short or long term. These effects may include symptoms such as emotional distress, disrupted routines such as sleeping and eating, feeling dazed or numb, avoiding reminders of the accident, distressing thoughts about the accident, grief, feeling on edge, or just not feeling like yourself. There are many different symptoms of psychological trauma. These symptoms, if persistent, can negatively affect an individual's social and working life, and significantly reduce quality of life as a whole.

Children

Children experience psychological trauma, too. Some symptoms of psychological trauma in children include bad dreams, showing elements of the accident in their play, and mood changes. They may seem to need more reassurance than usual. Note any changes in attitude or behaviour.

Psychological Intervention

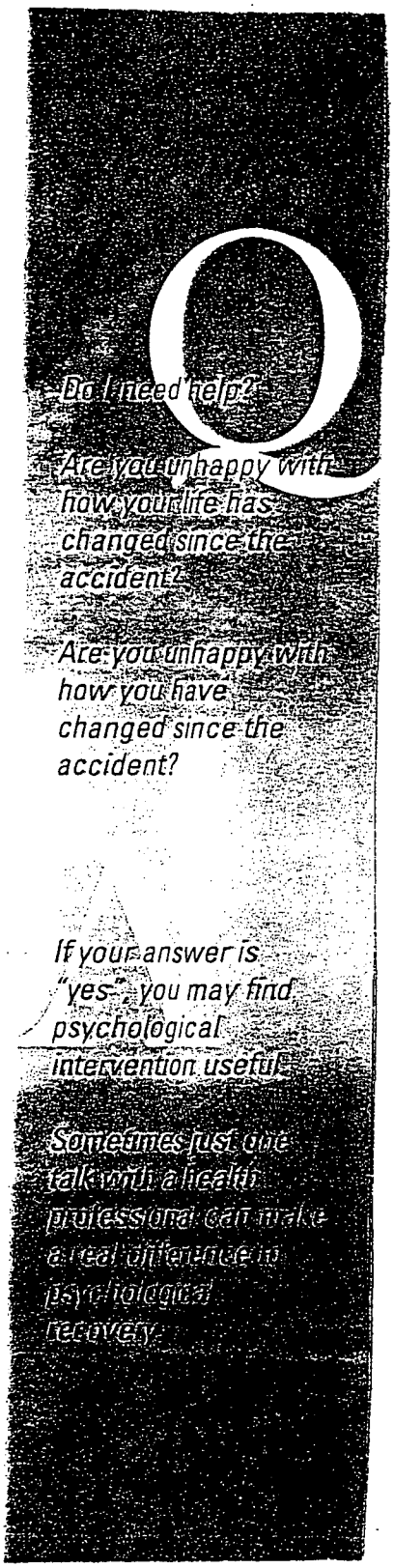
What is it?

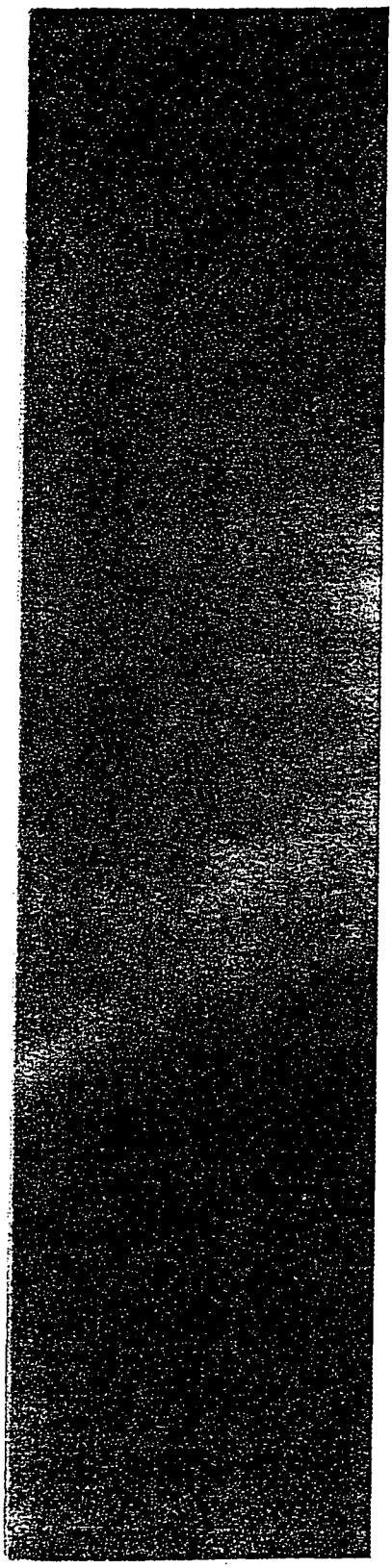
Psychological intervention is any professional service that caters for your psychological needs. This may be talking to your doctor, a psychologist, a psychiatrist, or other mental health professional.

Who should I go to?

Intervention can range from simply talking about the accident with someone who has professional knowledge of trauma, to more specific therapeutic treatments. There are many treatments for trauma.

Some involve medication, and others do not. Clinical psychologists specialising in trauma provide a range of non-medication based approaches. As a consumer, you have many choices regarding psychological intervention. Your GP will be able to advise you on the range of options available to you.





Notes for Survivors

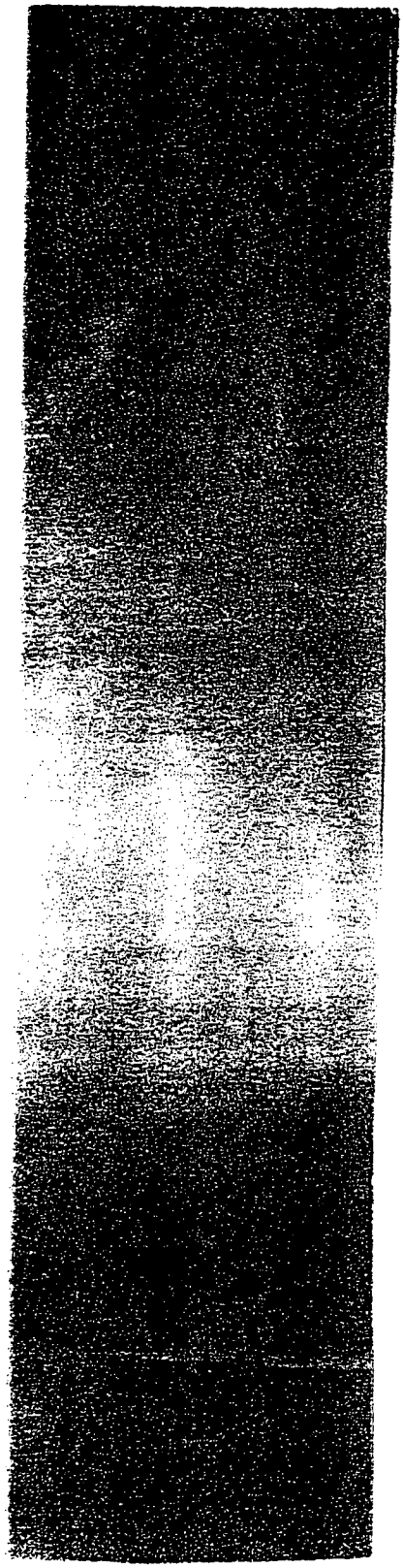
Posttraumatic psychological symptoms can keep your body under a high level of stress long after the event. Not all strategies may apply to you or your lifestyle, but to reduce your stress level following your accident, here are some suggestions to consider:

1. Physical exercise
2. Relaxation exercises
3. Talk about what happened and how you feel with someone you trust
4. Avoid alcohol and non-prescription drugs, and reduce cigarette and caffeine intake.
5. Try to maintain normal routines, but do not push yourself.
6. Participate in activities that you enjoy, and help you to feel good about yourself.
7. Keep a diary or write about the experience if you do not feel like talking to someone.
8. Spend quality time with family and friends.
9. Accept that trauma takes time to recover from, and that trauma symptoms are a natural response to a traumatic event.
10. Try to use the experience to cope better with the minor stresses or everyday life. You may have a new, positive perspective on the value of life.

Notes for Survivor's Support People

Your support may play an important role in helping the survivor to recover psychologically from the accident. Here are some suggestions to consider if you are having difficulty knowing what to do to help:

1. Offer support.
2. When the survivor wants to talk, listen without interrupting and do not be afraid of the survivor expressing their emotions.
3. Avoid trying to minimise what has happened or always trying to make the survivor look at the positive side.
4. Avoid encouraging the use of alcohol or non-prescription drugs.
5. Encourage the survivor to consider the suggestions on the previous page.
6. Help with everyday tasks to lighten the load.
7. Maximise the support network of the survivor by encouraging family and friends to keep in touch with the survivor.
8. Spend quality time with the survivor and reassure them that they are safe.
9. Encourage the survivor to seek psychological intervention if you are concerned about them.
10. Do not hesitate to seek counselling for yourself, as the support role can be difficult.



Where to go for Help

If you would like to talk to someone about your accident, or seek advice about psychological symptoms of trauma, there are various options available to you. Here are some suggestions :

Local GP
Psychologist
Psychiatrist
Counsellor

Check the Yellow Pages for further details